Inventor search history

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=> d his L110

     (FILE 'HCAPLUS' ENTERED AT 10:48:08 ON 21 NOV 2007)
L110         43 S L104 OR L106 OR L109

=> d que L110
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                 VIEW/DT
L101        144 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("STRITTMATTER S M"/AU OR
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                 WEI"/AU OR "LI WEI"/AU OR "LI W W"/AU OR "LI WEI W"/AU
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L105       5038 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L101 OR L102 OR L103)
L106         14 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L105 AND BIOGEN?/CO,CS,PA,SO
L107         97 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L105 AND YALE?/CO,CS,PA,SO
L108         78 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L107 AND L99
L109         30 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L108 AND (ALZHEIMER? OR
                 ALSHEIMER? OR AMYLOID? OR PLAQUE? OR NOGO? OR NOGOR? OR NOGO1?
                 OR NOGOR1? OR NGR? OR NGO? OR NGR1?)
L110         43 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L104 OR L106 OR L109


=> d his L150

     (FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 11:40:06 ON 21 NOV 2007)
L150         24 S L147 OR L149
                SAVE TEMP L150 HA669MLIN/A

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L99              QUE  ABB=ON  PLU=ON  AY<2005 OR PY<2005 OR PRY<2005 OR RE
                 VIEW/DT
L101        144 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ("STRITTMATTER S M"/AU OR
                 "STRITTMATTER STEPHEN"/AU OR "STRITTMATTER STEPHEN M"/AU OR
                 "STRITTMATTER STEPHEN MARK"/AU OR "STRITTMATTER STEPHEN S"/AU)
L102        228 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "LEE DANIEL"?/AU OR "LEE
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L103       4680 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "LI WEIWEI"/AU OR "LI WEI
                 WEI"/AU OR "LI WEI"/AU OR "LI W W"/AU OR "LI WEI W"/AU
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L105       5038 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L101 OR L102 OR L103)
L106         14 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L105 AND BIOGEN?/CO,CS,PA,SO
L147          6 SEA L104
L148         27 SEA L106
L149         20 SEA L148 AND L99
L150         24 SEA L147 OR L149


=> dup rem L110 L150
FILE 'HCAPLUS' ENTERED AT 12:07:44 ON 21 NOV 2007
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PROCESSING COMPLETED FOR L110
PROCESSING COMPLETED FOR L150
L151         50 DUP REM L110 L150 (17 DUPLICATES REMOVED)
                ANSWERS '1-43' FROM FILE HCAPLUS
                ANSWERS '44-49' FROM FILE BIOSIS
                ANSWER '50' FROM FILE DRUGU
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Inventor search results

=> d L151 1-50 ibib ab

L151 ANSWER 1 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2006:152149 HCAPLUS Full-text
DOCUMENT NUMBER: 144:290750
TITLE: Alzheimer precursor protein interaction with the nogo-66 receptor reduces amyloid-β plaque deposition
AUTHOR(S): Park, James H.; Gimbel, David A.; GrandPre, Tadzia; Lee, Jung-Kil; Kim, Ji-Eun; Li, Weiwei; Lee, Daniel H. S.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2006), 26(5), 1386-1395
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Pathophysiol. hypotheses for Alzheimer's disease (AD) are centered on the role of the amyloid plaque Aβ peptide and the mechanism of its derivation from the amyloid precursor protein (APP). As part of the disease process, an aberrant axonal sprouting response is known to occur near Aβ deposits. A Nogo to Nogo-66 receptor (NgR) pathway contributes to determining the ability of adult CNS axons to extend after traumatic injuries. Here, we consider the potential role of NgR mechanisms in AD. Both Nogo and NgR are mislocalized in AD brain samples. APP phys. assocs. with the NgR. Overexpression of NgR decrease Aβ production in neuroblastoma culture, and targeted disruption of NgR expression increases transgenic mouse brain Aβ levels, Aβ plaque deposition, and dystrophic neurites. Infusion of a soluble NgR fragment reduces Aβ levels, amyloid plaque deposits, and dystrophic neurites in a mouse transgenic AD model. Changes in NgR level produce parallel changes in secreted APPα and Aβ, implicating NgR as a blocker of secretase processing of APP. The NgR provides a novel site for modifying the course of AD and highlights the role of axonal dysfunction in the disease.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 2 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2006:722181 HCAPLUS Full-text
DOCUMENT NUMBER: 145:267732
TITLE: The Nogo66 receptor pathway and CNS axon regeneration: new hopes for treating CNS injuries and neurodegeneration
AUTHOR(S): Lee, Daniel HS; Seamans, Katherine W.
CORPORATE SOURCE: Neurobiology, Biogen Idec, Inc., Cambridge, MA, 02142, USA
SOURCE: Expert Opinion on Therapeutic Patents (2006), 16(8), 1041-1050
CODEN: EOTPEG; ISSN: 1354-3776
PUBLISHER: Informa Healthcare
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The neuronal leucine-rich repeat Nogo66 receptor (NgR) interacts with the myelin proteins Nogo66, myelin associated glycoprotein and oligodendrocyte myelin glycoprotein to inhibit axon growth. Modulation of

these cell surface NgR-dependent interactions or the inhibitory intracellular signaling pathways may promote axon growth in the CNS after injury and present an attractive axon regeneration platform for treating CNS injuries or even neurodegenerative disorders. Multiple NgR antagonism approaches, including soluble NgR proteins, anti-NgR antibodies, a Nogo-derived antagonist peptide and NgR signal transduction modulators, have demonstrated striking efficacies in promoting functional recoveries in animal models of spinal cord injury, stroke and multiple sclerosis. This review summarizes the neurobiol. of the NgR pathway and the various drug discovery strategies that are specifically based on modulation of the myelin-NgR interaction.
REFERENCE COUNT: 102 THERE ARE 102 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 3 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2004:828320 HCAPLUS Full-text
DOCUMENT NUMBER: 141:311429
TITLE: A Neutralizing Anti-Nogo66 Receptor Monoclonal Antibody Reverses Inhibition of Neurite Outgrowth by Central Nervous System Myelin
AUTHOR(S): Li, Weiwei; Walus, Lee; Rabacchi, Sylvia A.; Jirik, Adrienna; Chang, Ernie; Schauer, Jessica; Zheng, Betty H.; Benedetti, Nancy J.; Liu, Betty P.; Choi, Eugene; Worley, Dane; Silvian, Laura; Mo, Wenjun; Mullen, Colleen; Yang, Weixing; Strittmatter, Stephen M.; Sah, Dinah W. Y.; Pepinsky, Blake; Lee, Daniel H. S.
CORPORATE SOURCE: Biogen Idec, Inc., Cambridge, MA, 02142, USA
SOURCE: J. Biol. Chem. (2004), 279(42), 43780-43788
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Nogo66 receptor (NgR1) is a neuronal, leucine-rich repeat (LRR) protein that binds three central nervous system (CNS) myelin proteins, Nogo, myelin-associated glycoprotein, and oligodendrocyte myelin glycoprotein, and mediates their inhibitory effects on neurite growth. Although the LRR domains on NgR1 are necessary for binding to the myelin proteins, the exact epitope(s) involved in ligand binding is unclear. Here we report the generation and detailed characterization of an anti-NgR1 monoclonal antibody, 7E11. The 7E11 monoclonal antibody blocks Nogo, myelin-associated glycoprotein, and oligodendrocyte myelin glycoprotein binding to NgR1 with IC50 values of 120, 14, and 4.5 nM, resp., and effectively promotes neurite outgrowth of P3 rat dorsal root ganglia neurons cultured on a CNS myelin substrate. Further, we have defined the mol. epitope of 7E11 to be DNAQLR located in the third LRR domain of rat NgR1. Our data demonstrate that anti-NgR1 antibodies recognizing this epitope, such as 7E11, can neutralize CNS myelin-dependent inhibition of neurite outgrowth. Thus, specific anti-NgR1 antibodies may represent a useful therapeutic approach for promoting CNS repair after injury.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 4 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2004:1056241 HCAPLUS Full-text
DOCUMENT NUMBER: 142:17932
TITLE: Blockade of nogo-66, myelin-associated glycoprotein, and oligodendrocyte myelin glycoprotein by soluble nogo-66 receptor promotes axonal sprouting and recovery after spinal injury

AUTHOR(S): Li, Shuxin; Liu, Betty P.; Budel, Stephane; Li, Mingwei; Ji, Benxiu; Walus, Lee; Li, Weiwei; Jirik, Adrienna; Rabacchi, Sylvia; Choi, Eugene; Worley, Dane; Sah, Dinah W. Y.; Pepinsky, Blake; Lee, Daniel; Relton, Jane; Strittmatter, Stephen M.
CORPORATE SOURCE: Departments of Neurology and Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2004), 24(46), 10511-10520
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The growth of injured axons in the adult mammalian CNS is limited after injury. Three myelin proteins, Nogo, MAG (myelin-associated glycoprotein), and OMgp (oligodendrocyte myelin glycoprotein), bind to the Nogo-66 receptor (NgR) and inhibit axonal growth in vitro. Transgenic or viral blockade of NgR function allows axonal sprouting in vivo. Here, we administered the soluble function-blocking NgR ectodomain [aa 27-310; NgR (310)ecto] to spinal-injured rats. Purified NgR(310)ecto-Fc protein was delivered intrathecally after mid-thoracic dorsal over-hemisection. Axonal sprouting of corticospinal and raphe spinal fibers in NgR(310)ecto-Fc-treated animals correlates with improved spinal cord elec. conduction and improved locomotion. The ability of soluble NgR(310)ecto to promote axon growth and locomotor recovery demonstrates a therapeutic potential for NgR antagonism in traumatic spinal cord injury.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 5 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2003:647445 HCAPLUS Full-text
DOCUMENT NUMBER: 139:212250
TITLE: α7 Nicotinic Acetylcholine Receptors Mediate β-Amyloid Peptide-induced Tau Protein Phosphorylation
AUTHOR(S): Wang, Hoau-Yan; Li, Weiwei; Benedetti, Nancy J.; Lee, Daniel H. S.
CORPORATE SOURCE: Biogen Inc., Cambridge, MA, 02142, USA
SOURCE: Journal of Biological Chemistry (2003), 278(34), 31547-31553
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The Alzheimer's disease pathogenic peptide, β-amyloid42 (Aβ42), induces tau protein phosphorylation. Because hyperphosphorylated tau is a consistent component of neurofibrillary tangles, a pathol. hallmark of Alzheimer's disease, we investigated the signaling mols. involved in Aβ42-induced tau phosphorylation. We show that Aβ42 elicited rapid and reversible tau protein phosphorylation on three proline-directed sites (Ser-202, Thr-181, and Thr-231) in systems enriched in α7 nicotinic acetylcholine receptors (α7nAChR) including serum-deprived human SK-N-MC neuroblastoma cells and hippocampal synaptosomes. Although α7nAChR agonists induced similar phosphorylation, pretreatment with antisense-α7nAChR oligonucleotides (in cells) or α7nAChR antagonists (in cells and synaptosomes) attenuated Aβ-induced tau phosphorylation. Western analyses showed that the mitogen-activated kinase cascade proteins, ERKs and c-Jun N-terminal kinase (JNK-1), were concomitantly

activated by Aβ42, and their resp. kinase inhibitors suppressed Aβ-induced tau phosphorylation. More importantly, recombinant-activated ERKs and JNK-1 could differentially phosphorylate tau protein in vitro. Thus, the α7nAChR may mediate Aβ-induced tau protein phosphorylation via ERKs and JNK-1.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 6 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2003:922380 HCAPLUS Full-text
DOCUMENT NUMBER: 140:156523
TITLE: Targeting the NOGO receptor to threat central nervous system injuries
AUTHOR(S): Lee, Daniel H. S.; Strittmatter, Stephen M.; Sah, Dinah W. Y.
CORPORATE SOURCE: Biogen Inc., Cambridge, MA, 02142, USA
SOURCE: Nature Reviews Drug Discovery (2003), 2(11), 872-878
CODEN: NRDDAG; ISSN: 1474-1776
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Axonal damage is a key pathol. in many injuries of the central nervous system (CNS), such as spinal cord injury, traumatic brain injury and stroke, as well as in multiple sclerosis. An attractive drug discovery strategy to treat such conditions is to search for agents that promote CNS axonal regeneration. Historically, limited knowledge concerning the basis of poor CNS regeneration has precluded a rational drug discovery approach for promoting axonal regeneration. The recent identification of the Nogo receptor, which interacts with inhibitory myelin protein, established the crucial role of this mol. pathway in mediating the inhibitory effects of CNS myelin. This provides an unprecedented opportunity to manipulate adult CNS axonal regeneration. The development of therapeutics targeting the Nogo receptor has the potential to promote functional recovery and reverse the devastating consequences of CNS injuries.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 7 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2003:288472 HCAPLUS Full-text
DOCUMENT NUMBER: 139:5020
TITLE: Differential physiologic responses of α7 nicotinic acetylcholine receptors to β-amyloid1-40 and β-amyloid1-42
AUTHOR(S): Lee, Daniel H. S.; Wang, Hoau-Yan
CORPORATE SOURCE: Biogen Inc., Cambridge, MA, 02142, USA
SOURCE: Journal of Neurobiology (2003), 55(1), 25-30
CODEN: JNEUBZ; ISSN: 0022-3034
PUBLISHER: John Wiley & Sons, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The β-amyloid peptides (Aβ), Aβ1-40 and Aβ1-42, were implicated in Alzheimer's disease (AD) pathol. Although Aβ1-42 is generally considered to be the pathol. peptide in AD, both Aβ1-40 and Aβ1-42 were used in a variety of exptl. models without discrimination. Here the authors show that monomeric or oligomeric forms of the 2 Aβ peptides, when interact with the neuronal cation channel, α7 nicotinic acetylcholine receptors (α7nAChR), would result in distinct physiol. responses as measured by acetylcholine release and Ca influx expts. While Aβ1-42 effectively attenuated these α7nAChR-dependent physiol. to an extent that was apparently irreversible, Aβ1-40 showed a lower

inhibitory activity that could be restored upon washings with physiol. buffers or treatment with α7nAChR antagonists. These data suggest a clear pharmacol. distinction between Aβ1-40 and Aβ1-42.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 8 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:966580 HCAPLUS Full-text
DOCUMENT NUMBER: 147:315096
TITLE: Use of antagonists of the myelin-associated inhibitory factor receptor complex and neurotrophic factors for treatment of neurological diseases and disorders
INVENTOR(S): Lee, Daniel H. S.; Rossomando, Anthony; Weinreb, Paul H.
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA
SOURCE: PCT Int. Appl., 187pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007098283	A2	20070830	WO 2007-US5078	20070227

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2006-776657P P 20060227
US 2006-831459P P 20060718

AB Methods of promoting the survival, regeneration, and outgrowth of neurons in the treatment of neurol. disease are described. The methods involve use of antagonists of the MAIF (myelin-associated inhibitory factor) receptor complex in combination with neurotrophic factors. The receptor complex includes the Nogo receptor, the Sp35/LINGO protein, and the TAJ receptor (TNFRSF19.). These methods may be used to treat CNS disorders, stroke, or spinal injury.

L151 ANSWER 9 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:874243 HCAPLUS Full-text
DOCUMENT NUMBER: 147:269240
TITLE: Antagonists of the Nogo-1 receptor and their use in promoting neurite outgrowth in treatment of nerve injury
INVENTOR(S): Lee, Daniel H. S.; Wen, Dingyi; Pepinsky, Blake R.; Relton, Jane K.; Wang, Xinzhong; Lugovskoy, Alexey; Meier, Werner; Garber, Ellen A.; Silvian, Laura; Weinreb, Paul H.
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA
SOURCE: PCT Int. Appl., 190pp., which
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007089601	A2	20070809	WO 2007-US2199	20070126

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2006-762487P P 20060127
US 2006-831659P P 20060719

AB Peptides derived from the Nogo-1 receptor and antibodies to the receptor that can act as antagonists are described. These peptides and antibodies, and fusion proteins containing them, may be useful in promoting neurite outgrowth. The use of a Nogo receptor fusion protein with an Ig Fc domain to ameliorate spinal cord injury in rats is demonstrated.

L151 ANSWER 10 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:220128 HCAPLUS Full-text
DOCUMENT NUMBER: 146:302160
TITLE: Nogo receptor (NgR) disulfide structure, NgR signaling inhibiting NgR fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth
INVENTOR(S): Wen, Dingyi; Lee, Daniel H. S.; Pepinsky, R. Blake
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA
SOURCE: PCT Int. Appl., 89pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007025219	A2	20070301	WO 2006-US33369	20060825
WO 2007025219	A3	20070531		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,

GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: US 2005-710864P P 20050825

AB The present invention is directed to the use of certain polypeptides and polypeptide fragments of Nogo receptor-1 (NgR1) and Nogo receptor-2 (NgR2) for promoting neurite outgrowth, neuronal survival, and axonal regeneration in CNS neurons. Previous studies have shown that the entire leucine rich repeat (LRR) region of NgR1, including the C-terminal cap of LRR, LRR-CT, is needed for ligand binding, and that the adjacent CT stalk of the NgR1 contributes to interaction with its co-receptors. The inventors confirmed the amino acid sequence of human NgR1 by tryptic peptide mapping on a LC-MS system and determined the disulfide structure of NgR1 and NgR2 proteins from human and rat, particularly the LRR-CT regions. Typically, the polypeptides and polypeptide fragments of the invention act to block NgR-mediated inhibition of neuronal survival, neurite outgrowth or axonal regeneration of CNS (central nervous system) neurons by inhibiting signal transduction by the NgR complex.

L151 ANSWER 11 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1226364 HCAPLUS Full-text
DOCUMENT NUMBER: 146:26348
TITLE: Neuronal degeneration treatment with Nogo receptor antagonists
INVENTOR(S): Lee, Daniel H. S.; Sah, Dinah W. Y.; So, Kwok Fai; Wu, Wutian
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA; The University of Hong Kong
SOURCE: PCT Int. Appl., 49pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006124627	A2	20061123	WO 2006-US18484	20060512

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2005-679995P P 20050512
US 2005-735187P P 20051110

AB The disclosed invention provides methods for treating conditions of the eye involving death or degeneration of retinal ganglion cells, including glaucoma, by the administration of Nogo receptor-1 (NgR1) antagonists. The NgR1 antagonists comprise: a soluble form of NgR1 of different lengths and with different (up to 10) conservative amino acid substitutions; soluble NgR1 fusion with Ig Fc fragment; and different forms of anti-NgR1 antibodies and antibody fragments.

L151 ANSWER 12 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:411939 HCAPLUS Full-text
DOCUMENT NUMBER: 144:463018
TITLE: Receptor-binding Nogo-A peptides, Nogo receptor-1 mutant proteins with altered ligand binding, and pharmaceutical compositions
INVENTOR(S): Strittmatter, Stephen M.
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006047049	A2	20060504	WO 2005-US35719	20051003 <--
WO 2006047049	A3	20060601		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

AU 2005299974	A1	20060504	AU 2005-299974	20051003 <--
CA 2582581	A1	20060504	CA 2005-2582581	20051003 <--
EP 1805209	A2	20070711	EP 2005-851216	20051003 <--

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU

CN 101035803	A	20070912	CN 2005-80033350	20051003 <--
IN 2007DN02424	A	20070803	IN 2007-DN2424	20070330 <--

PRIORITY APPLN. INFO.: US 2004-615371P P 20041001 <--
WO 2005-US35719 W 20051003

AB Nogo, MAG, and OMgp are myelin-derived proteins that bind to a neuronal Nogo-66 Receptor (NgR) to limit axonal regeneration after CNS injury. Nogó-A protein may play the most prominent role in vivo, perhaps because its action is mediated both by NgR and by other receptors. Here, we extend our previous anal. of Nogo-A and NgR functional domains. In addition to a NgR-dependent Nogo-66 inhibitory domain and a NgR-independent Amino-Nogo-A specific domain, we identify a third Nogo-A specific domain that binds to NgR with nanomolar affinity. This third domain of 19 amino acids (aa) does not alter cell spreading or axonal outgrowth. Ala-scanning mutagenesis of surface residues in NgR partially distinguishes ligand binding sites for the two Nogo domains and for MAG, OMgp and Lingo-1. Fusion of the two NgR-binding Nogo-A domains creates a ligand with ten-fold enhanced affinity for NgR and converts a NgR antagonist peptide to an agonist. Thus, inhibition of axonal regeneration by NgR occurs after binding a subnanomolar bipartite Nogo-A ligand at a site partly overlapping with that for MAG and OMgp.

L151 ANSWER 13 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1127901 HCAPLUS Full-text

DOCUMENT NUMBER: 146:355078
TITLE: Extracellular regulators of axonal growth in the adult central nervous system
AUTHOR(S): Liu, Betty P.; Cafferty, William B. J.; Budel, Stephane O.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Philosophical Transactions of the Royal Society, B: Biological Sciences (2006), 361(1473), 1593-1610
CODEN: PTRBAE; ISSN: 0962-8436
PUBLISHER: Royal Society
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Robust axonal growth is required during development to establish neuronal connectivity. However, stable fiber patterns are necessary to maintain adult mammalian central nervous system (CNS) function. After adult CNS injury, factors that maintain axonal stability limit the recovery of function. Extracellular mols. play an important role in preserving the stability of the adult CNS axons and in restricting recovery from pathol. damage. Adult axonal growth inhibitors include a group of proteins on the oligodendrocyte, Nogo-A, myelin-associated glycoprotein, oligodendrocyte-myelin glycoprotein and ephrin-B3, which interact with axonal receptors, such as NgR1 and EphA4. Extracellular proteoglycans containing chondroitin sulfates also inhibit axonal sprouting in the adult CNS, particularly at the sites of astroglial scar formation. Therapeutic perturbations of these extracellular axonal growth inhibitors and their receptors or signalling mechanisms provide a degree of axonal sprouting and regeneration in the adult CNS. After CNS injury, such interventions support a partial return of neurol. function.

REFERENCE COUNT: 215 THERE ARE 215 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 14 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1054881 HCAPLUS Full-text
DOCUMENT NUMBER: 146:248381
TITLE: Axonal regeneration and recovery from chronic central nervous system injury
AUTHOR(S): Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University of School of Medicine, New Haven, CT, USA
SOURCE: Principles of Molecular Medicine (2nd Edition) (2006), 1165-1172. Editor(s): Runge, Marschall S.; Patterson, Cam. Humana Press Inc.: Totowa, N. J.
CODEN: 69IMWX; ISBN: 1-58829-202-9
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English

AB A review. Damage to the adult brain or spinal cord commonly produces persistent dysfunction without recovery. To replace lost neurons, stem cells, trophic factors, and transplantation of neural-competent cells might be relevant. Treatment of dysfunction based on the disconnection of surviving neurons requires the axonal regeneration from remaining neurons and a degree of plasticity in neuronal connectivity. Those neurol. conditions in which axonal regeneration and plasticity are most relevant are reviewed here. Recent scientific advances are likely to lead to the development of a novel group of therapeutics targeting axonal regeneration for the recovery of function in chronic neurol. dysfunction.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 15 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:823596 HCAPLUS Full-text
DOCUMENT NUMBER: 143:222540
TITLE: Treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists
INVENTOR(S): Relton, Jane K.; Engber, Thomas M.; Strittmatter, Stephen M.
PATENT ASSIGNEE(S): Biogen Idec MA Inc., USA; Yale University
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005074972	A2	20050818	WO 2005-US2535	20050128 <--
WO 2005074972	A3	20051222		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005210621	A1	20050818	AU 2005-210621	20050128 <--
CA 2555018	A1	20050818	CA 2005-2555018	20050128 <--
EP 1713494	A2	20061025	EP 2005-712127	20050128 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
CN 1946418	A	20070411	CN 2005-80009242	20050128 <--
BR 2005007272	A	20070626	BR 2005-7272	20050128 <--
JP 2007519737	T	20070719	JP 2006-551456	20050128 <--
MX 2006PA08392	A	20061030	MX 2006-PA8392	20060725 <--
IN 2006DN04365	A	20070831	IN 2006-DN4365	20060728 <--
KR 2007052237	A	20070521	KR 2006-717342	20060828 <--
PRIORITY APPLN. INFO.:			US 2004-540798P P	20040130 <--
			WO 2005-US2535 W	20050128

AB The invention provides methods for promoting regeneration or survival of dopaminergic neurons in a mammal displaying signs or symptoms of dopaminergic neuronal degeneration, including a human with Parkinson's disease, using Nogo receptor antagonists. The number of surviving dopaminergic neurons in the substantia nigra was significantly greater in Nogo receptor knockout mice compared to their heterozygote and wild-type litter-mate controls 4 wk after unilateral 6-hydroxydopamine injections. In addition, rotational behavior in response to apomorphine challenge was significantly lower in Nogo receptor-null mice. These data show increased neuronal survival and improved recovery of function in dopaminergic pathways in the brain after injury in mice lacking Nogo receptor. Treatment with the Nogo receptor antagonist sNgR(310)-Fc (soluble mature Nogo receptor fused with an Ig Fc fragment) increases cell survival and improved recovery in dopaminergic pathways in rat brain after injury. Thus, Nogo receptor antagonists comprising soluble Nogo receptor polypeptides, antibodies to the Nogo receptor protein, and small mol. may

promote regeneration and survival of dopaminergic neurons in mammals displaying degeneration.

L151 ANSWER 16 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:1250915 HCAPLUS Full-text
DOCUMENT NUMBER: 144:46919
TITLE: Disulfide Structure of the Leucine-Rich Repeat C-Terminal Cap and C-Terminal Stalk Region of Nogo-66 Receptor
AUTHOR(S): Wen, Dingyi; Wildes, Craig P.; Silvian, Laura; Walus, Lee; Mi, Sha; Lee, Daniel H. S.; Meier, Werner; Pepinsky, R. Blake
CORPORATE SOURCE: Biogenidec, Inc., Cambridge, MA, 02142, USA
SOURCE: Biochemistry (2005), 44(50), 16491-16501
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nogo-66 receptor (NgR1) is a leucine-rich repeat (LRR) protein that forms part of a signaling complex modulating axon regeneration. Previous studies have shown that the entire LRR region of NgR1, including the C-terminal cap of the LRR, LRRCT, is needed for ligand binding, and that the adjacent C-terminal region (CT stalk) of the NgR1 contributes to interaction with its coreceptors. To provide structure-based information for these interactions, we analyzed the disulfide structure of full-length NgR1. Our anal. revealed a novel disulfide structure in the C-terminal region of the NgR1, wherein the two Cys residues, Cys-335 and Cys-336, in the CT stalk are disulfide-linked to Cys-266 and Cys-309 in the LRRCT region: Cys-266 is linked to Cys-335, and Cys-309 to Cys-336. The other two Cys residues, Cys-264 and Cys-287, in the LRRCT region are disulfide-linked to each other. The anal. also showed that Cys-419 and Cys-429, in the CT stalk region, are linked to each other by a disulfide bond. Although published crystal structures of a recombinant fragment of NgR1 had revealed a disulfide linkage between Cys-266 and Cys-309 in the LRRCT region and we verified its presence in the corresponding fragment, this is artificially caused by the truncation of the protein, since this linkage was not detected in intact NgR1 or a slightly larger fragment containing Cys-335 and Cys-336. A structural model of the LRRCT with extended residues 311-344 from the CT stalk region is proposed, and its function in coreceptor binding is discussed.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 17 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:117342 HCAPLUS Full-text
DOCUMENT NUMBER: 143:52639
TITLE: Promoting the regeneration of axons within the central nervous system
AUTHOR(S): Park, James H.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, USA
SOURCE: From Neuroscience to Neurology (2005), 433-444.
Editor(s): Waxman, Stephen. Elsevier Inc.: Burlington, Mass.
CODEN: 69GMI9; ISBN: 0-12-738903-2
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English

AB A review. The peripheral nervous system axons, in contrast to the central nervous system, maintain their plasticity beyond the development of phase and remain capable of axonal regeneration after spinal cord injury. Progress in

identifying the mol. determinants promoting and inhibiting CNS axonal regeneration is discussed. Understanding how these determinants function, pharmacol. agents can be screened and medical treatments can be devised for the new therapeutic modality of axon regeneration in neuro-recovery.

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 18 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:927061 HCAPLUS Full-text
DOCUMENT NUMBER: 141:406109
TITLE: Treatment of conditions involving amyloid plaques
INVENTOR(S): Strittmatter, Stephen M.; Lee, Daniel H. S.; Li, Weiwei
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004093893	A2	20041104	WO 2004-US11728	20040416
WO 2004093893	A3	20050303		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004231742	A2	20041104	AU 2004-231742	20040416
AU 2004231742	A1	20041104		
CA 2522649	A1	20041104	CA 2004-2522649	20040416
EP 1615654	A2	20060118	EP 2004-759905	20040416
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
BR 2004009562	A	20060418	BR 2004-9562	20040416
CN 1832752	A	20060913	CN 2004-80016919	20040416
JP 2006523708	T	20061019	JP 2006-510107	20040416
MX 2005PA11100	A	20060418	MX 2005-PA11100	20051014
IN 2005DN04897	A	20070817	IN 2005-DN4897	20051026
NO 2005005392	A	20051115	NO 2005-5392	20051115
US 2007065429	A1	20070322	US 2006-553669	20060809

PRIORITY APPLN. INFO.: US 2003-463424P P 20030416
WO 2004-US11728 W 20040416

AB The invention provides methods for treating diseases involving aberrant amyloid-β (Aβ) peptide deposition, including Alzheimer's Disease, by the administration of Nogo receptor antagonists. The invention also provides method for reducing levels of Aβ peptide in a mammal by the administration of soluble Nogo receptor polypeptides.

L151 ANSWER 19 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:824033 HCAPLUS Full-text

DOCUMENT NUMBER: 141:290091
TITLE: Protein and cDNA sequences of a novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases
INVENTOR(S): Mi, Sha; McCoy, John; Pepinsky, R. Blake; Lee, Daniel H. S.
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085648	A2	20041007	WO 2004-US8323	20040317
WO 2004085648	A3	20041118		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004223464	A2	20041007	AU 2004-223464	20040317
AU 2004223464	A1	20041007		
CA 2519227	A1	20041007	CA 2004-2519227	20040317
EP 1606409	A2	20051221	EP 2004-757823	20040317
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
BR 2004008501	A	20060314	BR 2004-8501	20040317
CN 1798840	A	20060705	CN 2004-80013836	20040317
JP 2007524370	T	20070830	JP 2006-507330	20040317
IN 2005DN04139	A	20070831	IN 2005-DN4139	20050914
US 2007059793	A1	20070315	US 2005-553685	20051017
NO 2005004836	A	20051019	NO 2005-4836	20051019
PRIORITY APPLN. INFO.:			US 2003-455756P P	20030319
			US 2003-480241P P	20030620
			US 2003-492057P P	20030801
			WO 2004-US8323 W	20040317

AB The invention provides Sp35 polypeptides and fusion proteins thereof, Sp35 antibodies and antigen-binding fragments thereof and nucleic acids encoding the same. The invention also provides compns. comprising, and methods for making and using, such Sp35 antibodies, antigen-binding fragments thereof, Sp35 polypeptides and fusion proteins thereof.

L151 ANSWER 20 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:718635 HCAPLUS Full-text
DOCUMENT NUMBER: 141:236683
TITLE: Regeneration associated genes (RAGs) polypeptides, nucleic acids, and their use in related neuronal disease treatment and drug screening
INVENTOR(S): Strittmatter, Stephen S.
PATENT ASSIGNEE(S): Yale University, USA

SOURCE: PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004074433	A2	20040902	WO 2004-US2758	20040130 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2006127397	A1	20060615	US 2005-194074	20050729 <--
PRIORITY APPLN. INFO.:			US 2003-443485P P	20030130 <--
			WO 2004-US2758 A1	20040130 <--

AB The present invention relates generally to regeneration associated genes (RAGs). Specifically provided are 281-RAGs up-regulated mols. identified by microarray studies of L3-5 DRG (dorsal root ganglia) neurons one week after ipsilateral sciatic nerve transection. The significant upregulation of four RAGs: myosin-X, SOX11, FLRT3, Fn14, is demonstrated. The overexpression of Fn14, a receptor for tumor necrosis-like weak inducer of apoptosis (TWEAK), promotes neurite extension and growth cone formation in PC12 cells. Fn14 phys. interacts with the Rho family GTPase Rac1, and Rac1 is necessary for the Fn14-induced neuronal cell effects. Furthermore, the invention relates to structure-based methods and compns. useful in designing, identifying, and producing mols. which act as functional modulators of RAGs and RAG polypeptides. The invention further relates to methods of detecting, preventing, and treating RAG-associated disorders. The RAG ID NOs: 1-281 were not made available in the release of this patent.

L151 ANSWER 21 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:142908 HCAPLUS Full-text
DOCUMENT NUMBER: 140:198086
TITLE: Nogo receptor antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury
INVENTOR(S): Lee, Daniel H. S.; Pepinsky, R. Blake; Li, Weiwei; Rabacchi, Sylvia A.; Relton, Jane K.; Worley, Dane S.; Strittmatter, Stephen M. ; Sah, Dinah Y. W.
PATENT ASSIGNEE(S): Yale University, USA; Biogen, Inc.
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004014311	A2	20040219	WO 2003-US25004	20030807 <--
WO 2004014311	A3	20040429		

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CA 2495121		A1	20040219	CA 2003-2495121	20030807 <--
AU 2003264033		A1	20040225	AU 2003-264033	20030807 <--
EP 1534736		A2	20050601	EP 2003-785123	20030807 <--
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CN 1681838		A	20051012	CN 2003-821409	20030807 <--
JP 2005535329		T	20051124	JP 2004-527960	20030807 <--
BR 2003013331		A	20070724	BR 2003-13331	20030807 <--
AU 2004264405		A1	20050224	AU 2004-264405	20040130 <--
CA 2535007		A1	20050224	CA 2004-2535007	20040130 <--
WO 2005016955		A2	20050224	WO 2004-US2702	20040130 <--
WO 2005016955		A3	20060720		
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EP 1660517		A2	20060531	EP 2004-707073	20040130 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2004013426		A	20061017	BR 2004-13426	20040130 <--
JP 2007501612		T	20070201	JP 2006-522535	20040130 <--
CN 1926147		A	20070307	CN 2004-80029412	20040130 <--
NO 2005000685		A	20050510	NO 2005-685	20050209 <--
MX 2005PA01615		A	20050819	MX 2005-PA1615	20050210 <--
US 2005271655		A1	20051208	US 2005-55163	20050210 <--
IN 2005KN00382		A	20060512	IN 2005-KN382	20050309 <--
MX 2006PA01444		A	20060515	MX 2006-PA1444	20060203 <--
NO 2006001081		A	20060418	NO 2006-1081	20060306 <--
IN 2006DN01161		A	20070810	IN 2006-DN1161	20060306 <--
PRIORITY APPLN. INFO.:				US 2002-402866P	P 20020810 <--
				WO 2003-US25004	W 20030807 <--
				WO 2003-US325004	A 20030807 <--
				WO 2004-US2702	W 20040130 <--

AB Disclosed are immunogenic Nogo receptor-1 polypeptides, Nogo receptor-1 antibodies, antigen-binding fragments thereof, soluble Nogo receptors and fusion proteins thereof and nucleic acids encoding the same. Also disclosed are compns. comprising, and methods for making and using, such Nogo receptor antibodies, antigen-binding fragments, humanized and chimeric antibodies thereof, soluble Nogo receptors and fusion proteins thereof and nucleic acids or viral vector encoding the same for gene therapy. These Nogo receptor-1, antagonists are useful for inhibiting growth cone collapse of neuron, decreasing inhibition of neurite outgrowth, promoting survival of CNS neuron and axonal growth, and are therefore useful for treating multiple sclerosis,

ALS, Huntington's disease, Alzheimer's disease, Parkinson's disease, diabetes neuropathy, stroke, traumatic brain injury or spinal cord injury.

L151 ANSWER 22 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:20812 HCAPLUS Full-text
DOCUMENT NUMBER: 140:87723
TITLE: Modulators and modulation of the interacton between RGM and neogenin
INVENTOR(S): Strittmatter, Stephen; Mueller, Bernhard; Deitinghoff, Lutz
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004003150	A2	20040108	WO 2003-US20147	20030626 <--
WO 2004003150	A3	20040826		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2542171	A1	20040108	CA 2003-2542171	20030626 <--
AU 2003280420	A1	20040119	AU 2003-280420	20030626 <--
US 2006252101	A1	20061109	US 2005-519132	20050914 <--
PRIORITY APPLN. INFO.:			US 2002-392062P	P 20020626 <--
			WO 2003-US20147	W 20030626 <--

AB This invention relates to drug screening using mammalian repulsive guidance mols. (RGM's) and mammalian Neogenin. In addition, the invention provides for methods of preventing, alleviating or treating various disorders of the nervous system, angiogenic disorders or disorders of the cardio-vascular system and malignancies of different etiol. by disrupting the interaction between RGM and Neogenin.

L151 ANSWER 23 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:596882 HCAPLUS Full-text
DOCUMENT NUMBER: 141:168459
TITLE: Nogo receptor antagonism promotes stroke recovery by enhancing axonal plasticity
AUTHOR(S): Lee, Jung-Kil; Kim, Ji-Eun; Sivula, Michael; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2004), 24(27), 6209-6217
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal

LANGUAGE: English

AB After ischemic stroke, partial recovery of function frequently occurs and may depend on the plasticity of axonal connections. Here, we examine whether blockade of the Nogo-Nogo Receptor (NgR) pathway might enhance axonal sprouting and thereby recovery after focal brain infarction. Mutant mice lacking NgR or Nogo-AB recover complex motor function after stroke more completely than do control animals. After a stroke, greater nos. of axons emanating from the undamaged cortex cross the midline to innervate the contralateral red nucleus and the ipsilateral cervical spinal cord; this axonal plasticity is enhanced in ngr -/- or nogo-ab -/- mice. In rats with middle cerebral artery occlusion, both the recovery of motor skills and corticofugal axonal plasticity are promoted by intracerebroventricular administration of a function-blocking NgR fragment. Behavioral improvement occurs when therapy is initiated 1 wk after arterial occlusion. Thus, delayed pharmacol. blockade of the NgR promotes subacute stroke recovery by facilitating axonal plasticity.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 24 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:984215 HCAPLUS Full-text

DOCUMENT NUMBER: 141:377996

TITLE: Nogo-66 receptor prevents raphespinal and rubrospinal axon regeneration and limits functional recovery from spinal cord injury

AUTHOR(S): Kim, Ji-Eun; Liu, Betty P.; Park, James H.; Strittmatter, Stephen M.

CORPORATE SOURCE: Departments of Neurology and Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA

SOURCE: Neuron (2004), 44(3), 439-451
CODEN: NERNET; ISSN: 0896-6273

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Axon regeneration after injury to the adult mammalian CNS is limited in part by three inhibitory proteins in CNS myelin: Nogo-A, MAG, and OMgp. All three of these proteins bind to a Nogo-66 receptor (NgR) to inhibit axonal outgrowth in vitro. To explore the necessity of NgR for responses to myelin inhibitors and for restriction of axonal growth in the adult CNS, we generated ngr -/- mice. Mice lacking NgR are viable but display hypoactivity and motor impairment. DRG neurons lacking NgR do not bind Nogo-66, and their growth cones are not collapsed by Nogo -66. Recovery of motor function after dorsal hemisection or complete transection of the spinal cord is improved in the ngr -/- mice. While corticospinal fibers do not regenerate in mice lacking NgR , regeneration of some raphespinal and rubrospinal fibers does occur. Thus, NgR is partially responsible for limiting the regeneration of certain fiber systems in the adult CNS.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 25 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:263762 HCAPLUS Full-text

DOCUMENT NUMBER: 140:285004

TITLE: A new role for Nogo as a regulator of vascular remodeling

AUTHOR(S): Acevedo, Lisette; Yu, Jun; Erdjument-Bromage, Hediye; Miao, Robert Qing; Kim, Ji-Eun; Fulton, David; Tempst, Paul; Strittmatter, Stephen M.; Sessa, William C.

CORPORATE SOURCE: Boyer Center for Molecular Medicine, Department of Pharmacology and Program in Vascular Cell Signaling and Therapeutics, Yale University School of Medicine, New Haven, CT, 06536, USA

SOURCE: Nature Medicine (New York, NY, United States) (2004), 10(4), 382-388
CODEN: NAMEFI; ISSN: 1078-8956

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Although Nogo-A has been identified in the central nervous system as an inhibitor of axonal regeneration, the peripheral roles of Nogo isoforms remain virtually unknown. Here, using a proteomic anal. to identify proteins enriched in caveolae and/or lipid rafts (CEM/LR), we show that Nogo-B is highly expressed in cultured endothelial and smooth muscle cells, as well as in intact blood vessels. The N terminus of Nogo-B promotes the migration of endothelial cells but inhibits the migration of vascular smooth muscle (VSM) cells, processes necessary for vascular remodeling. Vascular injury in Nogo-A/B-deficient mice promotes exaggerated neointimal proliferation, and adenoviral-mediated gene transfer of Nogo-B rescues the abnormal vascular expansion in those knockout mice. Our discovery that Nogo-B is a regulator of vascular homeostasis and remodeling broadens the functional scope of this family of proteins.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 26 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:646415 HCAPLUS Full-text

DOCUMENT NUMBER: 141:330089

TITLE: Neonatal hypoxia suppresses oligodendrocyte Nogo-A and increases axonal sprouting in a rodent model for human prematurity

AUTHOR(S): Weiss, Jared; Takizawa, Bayan; McGee, Aaron; Stewart, William B.; Zhang, Heping; Ment, Laura; Schwartz, Michael; Strittmatter, Stephen

CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06520, USA

SOURCE: Experimental Neurology (2004), 189(1), 141-149
CODEN: EXNEAC; ISSN: 0014-4886

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Premature human infants frequently suffer from periventricular leukomalacia (PVL) characterized by the loss of central myelinated tracts in the brain [Neuropathol., 22 (2002) 193]. Rodent chronic sublethal hypoxia (CSH) from P3 to 33 (postnatal day 3-33) provides a model for PVL characterized by cerebral ventriculomegaly and redns. in cerebral white matter volume [Brain Res. Dev. Brain Res. 111 (1998) 197; Proc. Natl. Acad. Sci. USA 100 (2003) 11718]. Here, the authors demonstrate that mice exposed to CSH from P3 to P33 followed by normoxia from P33 to P75 continue to exhibit a locomotor hyperactivity that resembles behavioral changes observed in some human children with very low birth wts. Because periventricular white matter is specifically lost in PVL, the authors examined the expression of oligodendrocyte proteins. Hypoxic rearing dramatically decreases the level of the axon outgrowth inhibitor Nogo-A in oligodendrocytes of CNS white matter at P12. The Nogo-A decrease exceeds the moderate decrease in another myelin protein, myelin associated glycoprotein (MAG). Although myelin protein expression returns to normal by maturity (P75), persistent abnormalities in axonal trajectories are detectable. Anterograde axonal tracing from motor cortex demonstrates ectopic

corticofugal fibers in the corticospinal tract (CST), corpus callosum, and caudate nucleus of adult animals reared in CSH. Thus, hypoxia-induced reduction in myelin-derived axon outgrowth inhibitors appears to contribute axonal misconnection to the pathol. of very low birth weight infants.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 27 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:416397 HCAPLUS Full-text
DOCUMENT NUMBER: 139:332941
TITLE: Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal cord injury
AUTHOR(S): Li, Shuxin; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Journal of Neuroscience (2003), 23(10), 4219-4227
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Traumatized axons possess an extremely limited ability to regenerate within the adult mammalian CNS. The myelin-derived axon outgrowth inhibitors Nogo, oligodendrocyte-myelin glycoprotein, and myelin-associated glycoprotein, all bind to an axonal Nogo-66 receptor (NgR) and at least partially account for this lack of CNS repair. Although the intrathecal application of an NgR competitive antagonist at the time of spinal cord hemisection induces significant regeneration of corticospinal axons, such immediate local therapy may not be as clin. feasible for cases of spinal cord injury. Here, we consider whether this approach can be adapted to systemic therapy in a postinjury therapeutic time window. S.C. treatment with the NgR antagonist peptide NEP1-40 (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth protein SPRR1A (small proline-rich repeat protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal cord injury is enhanced. Furthermore, delaying the initiation of systemic NEP1-40 administration for up to 1 wk after cord lesions does not limit the degree of axon sprouting and functional recovery. This indicates that the regenerative capacity of transected corticospinal tract axons persists for weeks after injury. Systemic Nogo-66 receptor antagonists have therapeutic potential for subacute CNS axonal injuries such as spinal cord trauma.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 28 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:222475 HCAPLUS Full-text
DOCUMENT NUMBER: 139:66787
TITLE: Rho kinase inhibition enhances axonal regeneration in the injured CNS
AUTHOR(S): Fournier, Alyson E.; Takizawa, Bayan T.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2003), 23(4), 1416-1423
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience

DOCUMENT TYPE: Journal
LANGUAGE: English

AB Myelin-associated inhibitors limit axonal regeneration in the injured brain and spinal cord. A common target of many neurite outgrowth inhibitors is the Rho family of small GTPases. Activation of Rho and a downstream effector of Rho, p160ROCK, inhibits neurite out-growth. Here, we demonstrate that Rho is directly activated by the myelin-associated inhibitor Nogo-66. Using a binding assay to measure Rho activity, we detected increased levels of GTP Rho in PC12 and dorsal root ganglion (DRG) cell lysates after Nogo-66 stimulation. Rho activity levels were not affected by Amino-Nogo stimulation. Rho inactivation with C3 transferase promotes neurite outgrowth of chick DRG neurons in vitro, but with the delivery method used here, it fails to promote neurite outgrowth after corticospinal tract (CST) lesions in the adult rat. Inhibition of p160ROCK with Y-27632 also promotes neurite outgrowth on myelin-associated inhibitors in vitro. Furthermore, Y-27632 enhances sprouting of CST fibers in vivo and accelerates locomotor recovery after CST lesions in adult rats.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 29 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:495446 HCAPLUS Full-text
DOCUMENT NUMBER: 140:3143
TITLE: Nogo-C is sufficient to delay nerve regeneration
AUTHOR(S): Kim, Ji-Eun; Bonilla, Iris E.; Qiu, Dike; Strittmatter, Stephen M.
CORPORATE SOURCE: Departments of Neurology and Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Molecular and Cellular Neuroscience (2003), 23(3), 451-459
CODEN: MOCNED; ISSN: 1044-7431
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Axonal regeneration succeeds in the peripheral but not central nervous system of adult mammals. Peripheral clearance of myelin coupled with selective CNS expression of axon growth inhibitors, such as Nogo , may account for this reparative disparity. To assess the sufficiency of Nogo for limiting axonal regeneration, the authors generated transgenic mice expressing Nogo-C in peripheral Schwann cells. Nogo-C includes the panisoform inhibitory Nogo-66 domain, but not a second Nogo-A-specific inhibitory domain, allowing a selective consideration of the Nogo-66 region. The oct-6::nogo-c transgenic mice regenerate axons less rapidly than do wild-type mice after mid-thigh sciatic nerve crush. The delayed axonal regeneration is associated with a decreased recovery rate for motor function after sciatic nerve injury. Thus, expression of the Nogo-66 domain by otherwise permissive myelinating cells is sufficient to hinder axonal reextension after trauma.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 30 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:269429 HCAPLUS Full-text
DOCUMENT NUMBER: 139:130999
TITLE: The Nogo-66 receptor: focusing myelin inhibition of axon regeneration
AUTHOR(S): McGee, Aaron W.; Strittmatter, Stephen M.
CORPORATE SOURCE: Departments of Neurology and Neurobiology, Yale University School of Medicine, New Haven,

CT, 06520, USA
SOURCE: Trends in Neurosciences (2003), 26(4), 193-198
CODEN: TNSCDR; ISSN: 0166-2236
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. CNS myelin inhibits axonal outgrowth in vitro and is one of several obstacles to functional recovery following spinal cord injury. Central to our current understanding of myelin-mediated inhibition are the membrane protein Nogo and the Nogo-66 receptor (NgR). New findings implicate NgR as a point of convergence in signal transduction for several myelin-associated inhibitors. Addnl. studies have identified a potential coreceptor for NgR as p75NTR, and a second-messenger pathway involving RhoA that inhibits neurite elongation. Although these findings expand our understanding of the mol. determinants of adult CNS axonal regrowth, the physiol. roles of myelin-associated inhibitors in the intact adult CNS remain ill-defined.

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 31 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:377761 HCAPLUS Full-text
DOCUMENT NUMBER: 139:115755
TITLE: Axon regeneration in young adult mice lacking Nogo-A/B
AUTHOR(S): Kim, Ji-Eun; Li, Shuxin; GrandPre, Tadzia; Qiu, Dike; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology Department of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Neuron (2003), 38(2), 187-199
CODEN: NERNET; ISSN: 0896-6273
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB After injury, axons of the adult mammalian brain and spinal cord exhibit little regeneration. It has been suggested that axon growth inhibitors, such as myelin-derived Nogo, prevent CNS axon repair. To investigate this hypothesis, we analyzed mice with a nogo mutation that eliminates Nogo-A/B expression. These mice are viable and exhibit normal locomotion. Corticospinal tract tracing reveals no abnormality in uninjured nogo-A/B-/- mice. After spinal cord injury, corticospinal axons of young adult nogo-A/B-/- mice sprout extensively rostral to a transection. Numerous fibers regenerate into distal cord segments of nogo-A/B-/- mice. Recovery of locomotor function is improved in these mice. Thus, Nogo-A plays a role in restricting axonal sprouting in the young adult CNS after injury.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 32 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276162 HCAPLUS Full-text
DOCUMENT NUMBER: 136:322700
TITLE: Sequence homologs of the Nogo receptor and their use as targets for control of axonal growth in the treatment of neurological disease
INVENTOR(S): Strittmatter, Stephen M.; Cate, Richard L.; Sah, Dinah W. Y.
PATENT ASSIGNEE(S): Yale University, USA; Biogen, Inc.
SOURCE: PCT Int. Appl., 277 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002029059	A2	20020411	WO 2001-US31488	20011006 <--
WO 2002029059	A3	20030123		
WO 2002029059	A9	20030515		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2424834	A1	20020411	CA 2001-2424834	20011006 <--
AU 200211539	A	20020415	AU 2002-11539	20011006 <--
US 2003124704	A1	20030703	US 2001-972546	20011006 <--
EP 1325130	A2	20030709	EP 2001-979595	20011006 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004528809	T	20040924	JP 2002-532629	20011006 <--
NZ 525422	A	20060929	NZ 2001-525422	20011006 <--
AU 2002211539	B2	20070125	AU 2002-211539	20011006 <--
US 2005048520	A1	20050303	US 2003-735256	20031212 <--
US 7173118	B2	20070206		
US 2007104713	A1	20070510	US 2006-544013	20061006 <--
PRIORITY APPLN. INFO.:			US 2000-238361P	P 20001006 <--
			US 2001-972546	B1 20011006 <--
			WO 2001-US31488	W 20011006 <--
			US 2003-735256	A3 20031212 <--

AB The invention relates generally to genes that encode proteins that inhibit axonal growth. The invention relates specifically to genes encoding NgR protein homologs in humans and mice. The invention also includes compns. and methods for modulating the expression and activity of Nogo and the NgR proteins. Specifically, the invention includes peptides, proteins and antibodies that block Nogo -mediated inhibition of axonal extension. The compns. and methods of the invention are useful in the treatment of cranial or cerebral trauma, spinal cord injury, stroke or a demyelinating disease. The homologs were identified by TBLASTN querying of human and mouse genomic sequence databases.

L151 ANSWER 33 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:849092 HCAPLUS Full-text
DOCUMENT NUMBER: 138:252149
TITLE: Truncated soluble Nogo receptor binds Nogo-66 and blocks inhibition of axon growth by myelin
AUTHOR(S): Fournier, Alyson E.; Gould, Graham C.; Liu, Betty P.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2002), 22(20), 8876-8883

CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB CNS myelin contains axon outgrowth inhibitors, such as Nogo, that restrict regenerative growth after injury. An understanding of the mechanism of Nogo signaling through its receptor (NgR) is critical to developing strategies for overcoming Nogo-mediated inhibition. Here we analyze the function of NgR domains in outgrowth inhibition. Anal. of alkaline phosphatase (AP)-Nogo binding in COS-7 cells reveals that the leucine-rich repeat domain is necessary and sufficient for Nogo binding and NgR multimerization. Viral infection of embryonic day 7 chick retinal ganglion cells with mutated NgR demonstrates that the NgR C-terminal domain is required for inhibitory signaling but not ligand binding. The NgR glycosylphosphatidylinositol domain is not essential for inhibitory signaling but may facilitate Nogo responses. From this anal., we have developed a soluble, truncated version of the Nogo receptor that antagonizes outgrowth inhibition on both myelin and Nogo substrates. These data suggest that NgR mediates a significant fraction of myelin inhibition of axon outgrowth.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 34 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:519542 HCAPLUS Full-text
DOCUMENT NUMBER: 137:260565
TITLE: Localization of Nogo-A and Nogo-66 receptor proteins at sites of axon-myelin and synaptic contact
AUTHOR(S): Wang, Xingxing; Chun, Soo-Jin; Treloar, Helen; Vartanian, Timothy; Greer, Charles A.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Neuroscience (2002), 22(13), 5505-5515
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Axon regeneration in the adult CNS is limited by the presence of inhibitory proteins. An interaction of Nogo on the oligodendrocyte surface with Nogo-66 Receptor (NgR) on axons has been suggested to play an important role in limiting axonal growth. Here, we compare the localization of these two proteins immunohistochem. as a test of this hypothesis. Throughout much of the adult CNS, Nogo-A is detected on oligodendrocyte processes surrounding myelinated axons, including areas of axon-oligodendrocyte contact. The NgR protein is detected selectively in neurons and is present throughout axons, indicating that Nogo-A and its receptor are juxtaposed along the course of myelinated fibers. NgR protein expression is restricted to postnatal neurons and their axons. In contrast, Nogo-A is observed in myelinating oligodendrocytes, embryonic muscle, and neurons, suggesting that Nogo-A has addnl. physiol. roles unrelated to NgR binding. After spinal cord injury, Nogo-A is upregulated to a moderate degree, whereas NgR levels are maintained at constant levels. Taken together, these data confirm the apposition of Nogo ligand and NgR receptor in situations of limited axonal regeneration and support the hypothesis that this system regulates CNS axonal plasticity and recovery from injury.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 35 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:647265 HCAPLUS Full-text
DOCUMENT NUMBER: 138:2689
TITLE: Myelin-associated glycoprotein as a functional ligand for the Nogo-66 receptor
AUTHOR(S): Liu, Betty P.; Fournier, Alyson; GrandPre, Tadzia; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Science (Washington, DC, United States) (2002), 297(5584), 1190-1193
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Axonal regeneration in the adult central nervous system (CNS) is limited by two proteins in myelin, Nogo and myelin-associated glycoprotein (MAG). The receptor for Nogo (NgR) has been identified as an axonal glycosyl-phosphatidyl-inositol (GPI)-anchored protein, whereas the MAG receptor has remained elusive. Here, we show that MAG binds directly, with high affinity, to NgR. Cleavage of GPI-linked proteins from axons protects growth cones from MAG-induced collapse, and dominant-neg. NgR eliminates MAG inhibition of neurite outgrowth. MAG-resistant embryonic neurons are rendered MAG-sensitive by expression of NgR. MAG and Nogo-66 activate NgR independently and serve as redundant NgR ligands that may limit axonal regeneration after CNS injury.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 36 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:403264 HCAPLUS Full-text
DOCUMENT NUMBER: 137:362909
TITLE: Nogo-66 receptor antagonist peptide promotes axonal regeneration
AUTHOR(S): GrandPre, Tadzia; Li, Shuxin; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Nature (London, United Kingdom) (2002), 417(6888), 547-551
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Myelin-derived axon outgrowth inhibitors, such as Nogo, may account for the lack of axonal regeneration in the central nervous system (CNS) after trauma in adult mammals. A 66-residue domain of Nogo (Nogo-66) is expressed on the surface of oligodendrocytes and can inhibit axonal outgrowth through an axonal Nogo-66 receptor (NgR). The IN-1 monoclonal antibody recognizes Nogo-A and promotes corticospinal tract regeneration and locomotor recovery; however, the undefined nature of the IN-1 epitope in Nogo, the limited specificity of IN-1 for Nogo, and nonspecific anti-myelin effects have prevented a firm conclusion about the role of Nogo-66 or NgR. Here, we identify competitive antagonists of NgR derived from amino-terminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (NEP1-40) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that NgR mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of NEP1-40 to rats with mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional

recovery. Thus, Nogo-66 and NgR have central roles in limiting axonal regeneration after CNS injury, and NEP1-40 provides a potential therapeutic agent.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 37 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:909304 HCAPLUS Full-text
DOCUMENT NUMBER: 138:300857
TITLE: Nogo and the Nogo-66 receptor
AUTHOR(S): Fournier, Alyson E.; GrandPre, Tadzia; Gould, Graham; Wang, Xingxing; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Progress in Brain Research (2002), 137(Spinal Cord Trauma), 361-369
CODEN: PBRRA4; ISSN: 0079-6123
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Nogo has been identified as a component of central nervous system (CNS) myelin preventing axonal regeneration in the adult vertebrate CNS. Our previous anal. of Nogo-A demonstrated that an axon-inhibiting 66 aa domain is expressed at the extracellular surface and the endoplasmic reticulum lumen of transfected cells and oligodendrocytes. We have identified a brain-specific, leucine-rich repeat protein with high affinity for soluble Nogo-66. Cleavage of the Nogo-66 receptor from axonal surfaces renders neurons insensitive to Nogo-66. Nogo-66 receptor expression is sufficient to impart Nogo-66 axonal inhibition to unresponsive neurons. With identified ligand and receptor components, structure-function determinants for inhibition of axon regeneration can now be mapped. The relative contribution of Nogo, myelin-associated glycoprotein, chondroitin sulfate proteoglycan and oligodendrocyte myelin glycoprotein to myelin inhibition can be assessed. Blockade of Nogo-66 interaction with its receptor provides one potential avenue to promote axonal regeneration after adult mammalian CNS injury.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 38 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:694987 HCAPLUS Full-text
DOCUMENT NUMBER: 137:350109
TITLE: Modulation of axonal regeneration in neurodegenerative disease. Focus on Nogo
AUTHOR(S): Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Molecular Neuroscience (2002), 19(1/2), 117-121
CODEN: JMNEES; ISSN: 0895-8696
PUBLISHER: Humana Press Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears to have a physiol. role in regulating structural plasticity. The possibility that the Nogo system contributes to pathol. and compensatory plasticity in Alzheimer's Disease is considered.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 39 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:526105 HCAPLUS Full-text
DOCUMENT NUMBER: 135:117242
TITLE: Protein and cDNA sequences of human and mouse Nogo receptors, and therapeutic uses thereof for diseases associated with Nogo receptor-mediated blockade of axonal growth
INVENTOR(S): Strittmatter, Stephen M.
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001051520	A2	20010719	WO 2001-US1041	20010112 <--
WO 2001051520	A3	20020418		
WO 2001051520	A9	20020718		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2397199	A1	20010719	CA 2001-2397199	20010112 <--
AU 200129401	A	20010724	AU 2001-29401	20010112 <--
AU 784349	B2	20060316		
EP 1248803	A2	20021016	EP 2001-942367	20010112 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001007613	A	20021119	BR 2001-7613	20010112 <--
HU 2002003863	A2	20030328	HU 2002-3863	20010112 <--
JP 2003519481	T	20030624	JP 2001-551104	20010112 <--
EE 200200386	A	20031215	EE 2002-386	20010112 <--
NZ 520065	A	20060224	NZ 2001-520065	20010112 <--
NZ 541694	A	20060831	NZ 2001-541694	20010112 <--
NZ 547791	A	20070427	NZ 1991-5477	20010112 <--
US 2002077295	A1	20020620	US 2001-972599	20011006 <--
US 7119165	B2	20061010		
IN 2002KN00890	A	20060922	IN 2002-KN890	20020703 <--
BG 106907	A	20030530	BG 2002-106907	20020705 <--
ZA 2002005403	A	20040120	ZA 2002-5403	20020705 <--
NO 2002003387	A	20020911	NO 2002-3387	20020712 <--
MX 2002PA06885	A	20040405	MX 2002-PA6885	20020712 <--
AU 2006200819	A1	20060323	AU 2006-200819	20060227 <--

PRIORITY APPLN. INFO.:			
US 2000-175707P	P	20000112 <--
US 2000-207366P	P	20000526 <--
US 2000-236378P	P	20000929 <--
AU 2001-29401	A3	20010112 <--
NZ 2001-541694	A3	20010112 <--
US 2001-758140	A2	20010112 <--
WO 2001-US1040	A2	20010112 <--

WO 2001-US1041 W 20010112 <--

AB The invention provides protein and cDNA sequences of human and mouse Nogo receptor proteins and biol. active Nogo (ligand) protein fragments, which are members of the reticulon family proteins. Also disclosed are compns. and methods for modulating the expression or activity of the Nogo and Nogo receptor protein. Also disclosed are peptides which block Nogo-mediated inhibition of axonal extension. The compns. and methods of the invention are useful in the treatment of cranial or cerebral trauma, spinal cord injury, stroke or a demyelinating disease.

L151 ANSWER 40 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:785101 HCAPLUS Full-text
DOCUMENT NUMBER: 136:67265
TITLE: Nogo: A molecular determinant of axonal growth and regeneration
AUTHOR(S): Tadzia, Grandpre; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Neuroscientist (2001), 7(5), 377-386
CODEN: NROSFJ; ISSN: 1073-8584
PUBLISHER: Sage Publications, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review and discussion with many refs. Following injury, axons of the adult mammalian central nervous system (CNS) fail to regenerate. As a result, CNS trauma generally results in severe and persistent functional deficits. The inability of CNS axons to regenerate is largely associated with nonneuronal aspects of the CNS environment that are inhibitory to axonal elongation. This inhibition is mediated by the glial scar, including reactive astrocytes, and by the myelin-associated neurite outgrowth inhibitors chondroitin sulfate proteoglycans, myelin-associated glyco-protein, and Nogo. Nogo is an integral membrane protein that localizes to CNS, but not peripheral nervous system, myelin. In vitro characterization of Nogo has demonstrated its function as a potent inhibitor of axon elongation. In vivo neutralization of Nogo activity results in enhanced axonal regeneration and functional recovery following CNS injury as well as increased plasticity in uninjured CNS fibers. These findings suggest that Nogo may be a major contributor to the nonpermissive nature of the CNS environment.

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 41 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:66960 HCAPLUS Full-text
DOCUMENT NUMBER: 134:205513
TITLE: Identification of a receptor mediating Nogo -66 inhibition of axonal regeneration
AUTHOR(S): Fournier, Alyson E.; GrandPre, Tadzia; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Nature (London) (2001), 409(6818), 341-346
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nogo has been identified as a component of the central nervous system (CNS) myelin that prevents axonal regeneration in the adult vertebrate CNS. Anal. of Nogo-A has shown that an axon-inhibiting domain of 66 amino acids is

expressed at cellular surface and at the endoplasmic reticulum lumen of transfected cells and oligodendrocytes. The acidic amino terminus of Nogo-A is detected at the cytosolic face of cellular membranes and may contribute to inhibition of axon regeneration at sites of oligodendrocyte injury. Here we show that the extracellular domain of Nogo (Nogo -66) inhibits axonal extension, but does not alter non-neuronal cell morphol. In contrast, a multivalent form of the N terminus of Nogo-A affects the morphol. of both neurons and other cell types. Here we identify a brain-specific, leucine-rich-repeat protein with high affinity for soluble Nogo-66. Cleavage of the Nogo-66 receptor and other glycophosphatidylinositol-linked proteins from axonal surfaces renders neurons insensitive to Nogo-66. Nogo -66 receptor expression is sufficient to impart Nogo-66 axonal inhibition to unresponsive neurons. Disruption of the interaction between Nogo-66 and its receptor provides the potential for enhanced recovery after human CNS injury.

L151 ANSWER 42 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:175671 HCAPLUS Full-text
DOCUMENT NUMBER: 134:234759
TITLE: Repulsive factors and axon regeneration in the CNS
AUTHOR(S): Fournier, Alyson E.; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Current Opinion in Neurobiology (2001), 11(1), 89-94
CODEN: COPUEN; ISSN: 0959-4388
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with 46 refs. During the past year, a major advance in the study of axon regeneration was the mol. cloning of Nogo. The expression of Nogo protein by central nervous system (CNS) myelin may be a major factor in the failure of CNS axon regeneration. The effect of disrupting Nogo-dependent axon inhibition can now be studied conclusively. In related work, immunization with a Nogo -containing CNS myelin preparation was shown to promote regeneration and dramatic functional recovery after spinal cord trauma.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 43 OF 50 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:97195 HCAPLUS Full-text
DOCUMENT NUMBER: 132:220197
TITLE: Identification of the Nogo inhibitor of axon regeneration as a Reticulon protein
AUTHOR(S): GrandPre, Tadzia; Nakamura, Fumio; Vartanian, Timothy; Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, USA
SOURCE: Nature (London) (2000), 403(6768), 439-444
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Adult mammalian axon regeneration is generally successful in the peripheral nervous system (PNS) but is dismally poor in the central nervous system (CNS). However, many classes of CNS axons can extend for long distances in peripheral nerve grafts. A comparison of myelin from the CNS and the PNS has revealed

that CNS white matter is selectively inhibitory for axonal outgrowth. Several components of CNS white matter, N135, NI250(Nogo) and MAG, that have inhibitory activity for axon extension have been described. The IN-1 antibody, which recognizes N135 and NI250(Nogo), allows moderate degrees of axonal regeneration and functional recovery after spinal cord injury. Here we identify Nogo as a member of the Reticulon family, Reticulon 4-A. Nogo is expressed by oligodendrocytes but not by Schwann cells, and assocs. primarily with the endoplasmic reticulum. A 66-residue lumenal/extracellular domain inhibits axonal extension and collapses dorsal root ganglion growth cones. In contrast to Nogo, Reticulon 1 and 3 are not expressed by oligodendrocytes, and the 66-residue lumenal/extracellular domains from Reticulon 1, 2 and 3 do not inhibit axonal regeneration. These data provide a mol. basis to assess the contribution of Nogo to the failure of axonal regeneration in the adult CNS.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L151 ANSWER 44 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 8

ACCESSION NUMBER: 2003:47271 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300047271
TITLE: Consistent immunohistochemical detection of intracellular beta-amyloid42 in pyramidal neurons of Alzheimer's disease entorhinal cortex.
AUTHOR(S): D'Andrea, Michael R.; Nagele, Robert G.; Wang, Hoau-Yan; Lee, Daniel H. S. [Reprint Author]
CORPORATE SOURCE: Biogen Inc., 14 Cambridge Center, Cambridge, MA, 02142, USA
daniel_lee@biogen.com
SOURCE: Neuroscience Letters, (November 29 2002) Vol. 333, No. 3, pp. 163-166. print.
ISSN: 0304-3940 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Jan 2003
Last Updated on STN: 15 Jan 2003

AB We compared the effects of three pretreatment immunohistochemical techniques (no pretreatment, pepsin predigestion and heat pretreatment (HEAT)) for detecting intracellular beta-amyloid42 (Abeta42) in pyramidal neurons of formalin-fixed Alzheimer's disease (AD) cortices (n=25). Although all three protocols immunostained Abeta42 in amyloid plaques using four commercially-obtained Abeta42 specific antibodies, only the HEAT protocol consistently detected prominent intracellular Abeta42 in pyramidal neurons. This suggests that the Abeta42 present in amyloid plaques may be structurally distinct from that located within the neurons perhaps due to differential binding proteins coupling or a consequence of formalin fixation. Detection of an abundant intracellular Abeta42 in neurons may provide alternate explanations for the origin of dense-core amyloid plaques in AD cortices other than the conventional chronic extracellular Abeta42 deposition hypothesis.

L151 ANSWER 45 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:191837 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400190211
TITLE: The Nogo66 receptor as a drug discovery target for promoting CNS axon regeneration.
AUTHOR(S): Lee, D. H. S. [Reprint Author]; Li, S.; Kim, J.-E.; Liu, B. P.; Li, W. [Reprint Author]; Li, M. [Reprint Author]; Ji, B. [Reprint Author]; Walus, L. [Reprint Author]; Jirik, A. [Reprint Author]; Rabacchi, S. [Reprint

Author]; Choi, E. [Reprint Author]; Silvian, L. [Reprint Author]; Thill, G. [Reprint Author]; Benedetti, N. J. [Reprint Author]; Schauer, J. [Reprint Author]; Zheng, B. [Reprint Author]; Chang, E. [Reprint Author]; Mi, S. [Reprint Author]; Shao, Z. [Reprint Author]; Lee, X. [Reprint Author]; Zhang, M. [Reprint Author]; Mullen, C. [Reprint Author]; Worley, D. [Reprint Author]; Cate, R. [Reprint Author]; McCoy, J. [Reprint Author]; Sah, D. W. Y. [Reprint Author]; Pepinsky, B. [Reprint Author]; Relton, J. [Reprint Author]; Strittmatter, S. M.
CORPORATE SOURCE: Biogen Inc., Cambridge, MA, USA
SOURCE: Journal of Neurochemistry, (February 2004) Vol. 88, No. Supplement 1, pp. 13. print.
Meeting Info.: 6th Biennial Meeting of the Asian-Pacific Society for Neurochemistry (APSN). Hong Kong, China. February 04-07, 2004. Asian-Pacific Society for Neurochemistry.
CODEN: JONRA9. ISSN: 0022-3042.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2004
Last Updated on STN: 7 Apr 2004

L151 ANSWER 46 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:293946 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300293946
TITLE: NEUTRALIZATION OF MYELIN - ASSOCIATED NOGO - A BY A NOGO RECEPTOR - Fc FUSION PROTEIN.
AUTHOR(S): Li, W. [Reprint Author]; Walus, L. [Reprint Author]; Jirik, A. [Reprint Author]; Pepinsky, B. [Reprint Author]; Sah, D. W. Y. [Reprint Author]; Lee, D. H. S. [Reprint Author]; Fournier, A.; Strittmatter, S.; Rabacchi, S. A. [Reprint Author]
CORPORATE SOURCE: BIOGEN, Inc., Cambridge, MA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 333.2. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003

AB Nogo A plays a major role in the inhibitory activity of CNS myelin on axonal regeneration after CNS injury. We generated a secreted recombinant Nogo receptor-Fc fusion protein (Ig-sNogoR) and evaluated its ability to interfere with the Nogo-Nogo receptor interaction. CHO cells were transfected with a plasmid construct encoding the 1-310 residues of the extracellular domain of rat Nogo receptor fused with the Fc and hinge from rat IgG1. The secreted protein product was purified on protein A-Sepharose and characterized by N-terminal sequencing, SDS-PAGE, and Western blot and ELISA using antibodies raised against the Nogo receptor. Ig-sNogoR inhibits 125I-Nogo66 binding to the Nogo receptor in a scintillation proximity assay system with an IC50 apprx100nM. In addition, we tested Ig-sNogoR as a potential antagonist of the inhibitory effects of NogoA on P4 rat DRG neurite outgrowth in vitro. In this

assay, Ig-sNogoR fully reverses the inhibitory effects of NogoA-containing CNS myelin in a dose-dependent manner, with maximal protection seen at apprx0.5 muM. Thus, the NogoR-Fc fusion protein disrupts the NogoA-NogoR interaction and promotes neurite growth in the presence of CNS myelin, further substantiating the role of Nogo and NogoR in axonal regeneration.

L151 ANSWER 47 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:293947 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300293947
TITLE: CHARACTERIZATION OF A MONOCLONAL ANTI - NOGO RECEPTOR ANTIBODY.
AUTHOR(S): Mullen, C. [Reprint Author]; Li, W. [Reprint Author]; Rabacchi, S. [Reprint Author]; Jirik, A. [Reprint Author]; Yang, W. [Reprint Author]; Crowell, T. [Reprint Author]; Gardner, H. [Reprint Author]; Walus, L. [Reprint Author]; Sandrock, A. W. [Reprint Author]; Sah, D. W. Y. [Reprint Author]; Pepinsky, B. [Reprint Author]; Lee, D. H. S. [Reprint Author]; Miklasz, S. [Reprint Author]
CORPORATE SOURCE: Neurodegeneration, Biogen and comma; Inc., Cambridge, MA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 333.3. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting) Conference; Abstract; (Meeting Abstract) Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jun 2003 Last Updated on STN: 25 Jun 2003

AB CNS axons do not regenerate after injury because CNS myelin contains molecules that negatively regulate CNS axonal regeneration. The neuronal GPI-anchored protein, Nogo receptor, mediates inhibitory effects of CNS myelin on axonal regeneration. Modulation of the interaction of Nogo receptor with its ligands in CNS myelin may promote axonal regeneration, thereby providing a novel therapeutic opportunity for treating neurodegenerative disorders including spinal cord injuries, traumatic brain injuries, stroke and multiple sclerosis. We have generated murine monoclonal anti-Nogo receptor antibodies using full length Nogo receptor expressed on cells as the antigen. Here, we report on the characterization of one monoclonal anti-Nogo receptor antibody, 1H2. 1H2 is an IgG1, recognizes the carboxyl region of the Nogo receptor distal to the leucine rich repeat motifs, and binds COS cells expressing Nogo receptor in FACS analysis. The applications of this antibody in immunoprecipitation experiments and in studying the expression profile of Nogo receptor in tissues by immunohistochemistry and Western analyses will be discussed.

L151 ANSWER 48 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:293944 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300293944
TITLE: CHARACTERIZATION OF AN ANTI - NOGO RECEPTOR FAB THAT DISRUPTS NOGOA/NOGO RECEPTOR INTERACTION.
AUTHOR(S): Choi, E. D. [Reprint Author]; Rabacchi, S. A. [Reprint Author]; Li, W. [Reprint Author]; Jirik, A. [Reprint Author]; Pepinsky, B. [Reprint Author]; Sah, D. W. Y.

[Reprint Author]; Lee, D. H. S. [Reprint Author]; Worley, D. S. [Reprint Author]
CORPORATE SOURCE: BIOGEN, Inc., Cambridge, MA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 333.1. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting) Conference; Abstract; (Meeting Abstract) Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jun 2003 Last Updated on STN: 25 Jun 2003

AB The lack of axonal regeneration in mammalian CNS is at least partly due to the interaction of the myelin-associated NogoA protein with its receptor(s). With the goal of disrupting the Nogo/Nogo receptor pathway to promote axonal regeneration, we used the MorphoSys Fab-phage display technology to identify monovalent human Fabs that would specifically recognize the rat Nogo receptor (NogoR) (Fournier et.al., Nature 409, 341-346, 2001) with high affinity. One such Fab, 2E10, was obtained by screening the MorphoSys HuCAL Fab-1 phage library on 293EBNA cells transiently transfected with full-length rat NogoR. 2E10 stains COS cells expressing NogoR and inhibits the binding of iodinated Nogo66 to the Nogo receptor in a scintillation proximity assay system. The 2E10 Fab also behaved as an antagonist of the Nogo-NogoR interaction in an in vitro neurite outgrowth assay using P4 rat DRGs. At concentrations of immobilized CNS myelin that resulted in an inhibition of neurite outgrowth by apprx50%, 2E10 completely restored neurite outgrowth to levels found in the absence of CNS myelin. These results suggest that the 2E10 Fab can disrupt the NogoA/NogoR inhibitory pathway and overcome the inhibitory effects of CNS myelin on neurite outgrowth, further supporting the role that this pathway plays in inhibiting CNS axonal regeneration. The 2E10 Fab will be converted into a human-rat chimaeric antibody to facilitate future in vivo studies.

L151 ANSWER 49 OF 50 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:269130 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300269130
TITLE: SIGNAL TRANSDUCTION EVENTS OF THE BETA-AMYLOID42 AND ALPHA7 NICOTINIC ACETYLCHOLINE RECEPTOR INTERACTION.
AUTHOR(S): Lee, D. H. S. [Reprint Author]; Wang, H. Y.
CORPORATE SOURCE: Neurobiology, Biogen Inc, Cambridge, MA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 91.11. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting) Conference; (Meeting Poster) Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jun 2003 Last Updated on STN: 11 Jun 2003

AB beta-Amyloid42 binds to the neuronal alpha7 nicotinic acetylcholine receptor (alpha7nAChR) and modulates a variety of neurophysiological processes relevant to memory and cognition such as calcium homeostasis, neurotransmitter release and choline uptake. Recent findings that the beta-amyloid42-alpha7nAChR

interaction may activate ERKs and even tau protein phosphorylation further emphasize the roles of these proteins in Alzheimer's disease. To study the signal transduction mechanisms associated with the beta-amyloid42-alpha7nAChR interaction, here we report that in serum-starved SK-N-MC cells expressing alpha7nAChR, within a short time interval of even seconds of exposure to soluble, non-fibrillar beta-amyloid42, the cellular inositol tris-phosphate levels were elevated in a dose-dependent manner. The optimal dose of 10 nM beta-amyloid42 resulted in apprx6-fold increase of cellular inositol tris-phosphates. This was accompanied by an exclusive recruitment of phospholipase C-gamma2 to the cytoplasmic signaling complex associated with alpha7nAChR as shown by co-immunoprecipitation and Western analyses. Blockade of the inositol tris-phosphate receptor and reduction of intracellular calcium by pharmacological agents significantly suppressed ERKs activation by beta-amyloid42. These results suggest that the early signaling event of the beta-amyloid42-alpha7nAChR interaction involves the inositol phosphate pathway that will eventually lead to the activation of ERKs.

L151 ANSWER 50 OF 50 DRUGU COPYRIGHT 2007 THE THOMSON CORP on STN
ACCESSION NUMBER: 2002-42730 DRUGU P Full-text
TITLE: Targeting intracellular Abeta42 for Alzheimer's disease drug discovery.
AUTHOR: D Andrea M R; Lee D H S; Wang H Y; Nagele R G
CORPORATE SOURCE: Johnson+Johnson; Biogen; Univ.New-York-City; Univ.New-Jersey-Med.+Dent.
LOCATION: Spring House, Pa., Cambridge, Mass., New York, N.Y.; Stratford, N.J., USA
SOURCE: Drug Dev.Res. (56, No. 2, 194-200, 2002) 1 Fig. 79 Ref.
CODEN: DDREDK ISSN: 0272-4391
AVAIL. OF DOC.: Drug Discovery, Johnson and Johnson Pharmaceutical Research and Development, Welsh and McKean Roads, Spring Hous, PA 19477, U.S.A. (e-mail: mdandrea@prdus.jnj.com).
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB Regardless of the intracellular effects of Abeta (either awry signal transduction or simple obstruction of cell transport activities), the work summarized in this review led to the proposal of the alpha7 nicotinic acetylcholine receptor as a drug target for early intervention in Alzheimer's disease. Based on an inside-out hypothesis of dens-core plaque formation, it can be predicted that blockade of exogenous Abeta42 from entering vulnerable pyramidal neurons in vivo will reduce intraneuronal Abeta42 accumulation. This will, in turn, result in prolonging neuron survival time and hence, slow down the degeneration process. (No EX).

Text search history

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=> d his L100

     (FILE 'HCAPLUS' ENTERED AT 10:48:08 ON 21 NOV 2007)
L100          23 S L98 AND L99

=> d que L100
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L74          39 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L61 AND L70
L76          43 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L73 OR L74
L77           3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L60 AND L61
L79       11765 SEA FILE=HCAPLUS ABB=ON  PLU=ON  AMYLOID/CT
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L81           7 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L79 AND L69
L82          10 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L79 AND L70
L83          12 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L81 OR L82
L84          44 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L83 OR L76
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L87           QUE  ABB=ON  PLU=ON  ((PEPT? OR POLYPEPT?)(3A)(AGON? OR A
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L88           4 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L84 AND L87
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L90        7129 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L79 AND L89
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                OG? OR CAT? OR PIG? OR RAT? OR MOUSE? OR MICE? OR HUMAN?
                OR MONKEY? OR PLACENT? OR MARSUP?)(3A)(BRAIN? OR CNS? OR
                (CENTRAL)(2A)(NERVOUS)))
L92           QUE  ABB=ON  PLU=ON  (REDUC? OR ADMINIST? OR TREAT? OR AL
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L97          45 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L93 OR L94 OR L95 OR L96)
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                VIEW/DT
L100         23 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L98 AND L99


=> d his L146

     (FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 11:40:06 ON 21 NOV 2007)
L146         23 S L145 OR L131

=> d que L146
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                OR NGR1?)(5A)(AGON? OR ANTAGON? OR RECEPT? OR PEPTID? OR
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L85           QUE  ABB=ON  PLU=ON  (RECEPT?(3A)(AGON? OR ANTAGON?))
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L89           QUE  ABB=ON  PLU=ON  ((ALPHA? OR ALFA? OR BETA?)(3A)(PEPT
                ID? OR POLYPEPT? OR PROTE?))
L91           QUE  ABB=ON  PLU=ON  ((MAMMAL? OR PRIMAT? OR RODENT? OR D
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                OR MONKEY? OR PLACENT? OR MARSUP?)(3A)(BRAIN? OR CNS? OR
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L92           QUE  ABB=ON  PLU=ON  (REDUC? OR ADMINIST? OR TREAT? OR AL
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                EDICAT? OR THERAP?)
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                VIEW/DT
L111      192138 SEA (ALZHEIMER? OR ALSHEIMER?)
L112       68782 SEA (AMYLOID?(4N)(PLAQUE? OR PEPTID? OR POLYPEPT? OR PROTEIN?))

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L116          17 SEA L113 AND L70
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L131          21 SEA L128 OR L129 OR L130
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L145           5 SEA L144 AND L99
L146          23 SEA L145 OR L131


=> dup rem L100 L146
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L152 36 DUP REM L100 L146 (10 DUPLICATES REMOVED)
ANSWERS '1-23' FROM FILE HCAPLUS
ANSWERS '24-29' FROM FILE MEDLINE
ANSWERS '30-32' FROM FILE BIOSIS
ANSWERS '33-36' FROM FILE EMBASE

Text search results

=> d L152 1-23 ibib ed abs hitind

L152 ANSWER 1 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2007:152708 HCAPLUS Full-text
DOCUMENT NUMBER: 146:292365
TITLE: Novel modulators of amyloid-β precursor protein processing
AUTHOR(S): Tang, Bor Luen; Liou, Yih Cherng
CORPORATE SOURCE: Department of Biochemistry, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore
SOURCE: Journal of Neurochemistry (2007), 100(2), 314-323
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ED Entered STN: 12 Feb 2007
AB A review. Proteolytic processing of the amyloid precursor protein (APP) is modulated by the action of enzymes α-, β- and γ-secretases, with the latter two mediating the amyloidogenic production of amyloid-β (Aβ). Cellular modulators of APP processing are well known from studies of genetic mutations (such as those found in APP and presenilins) or polymorphisms (such as the apolipoprotein E4 ε-allele) that predisposes an individual to early or late-onset Alzheimer's disease. In recent years, several classes of mol. with modulating functions in APP processing and Aβ secretion have emerged. These include the neuronal Munc-18 interacting proteins (Mints)/X11s, members of the reticulon family (RTN-3 and RTN-4/ Nogo-B), the Nogo-66 receptor (NgR), the peptidylprolyl isomerase Pin1 and the Rho family GTPases and their effectors. Mints and NgR bind to APP directly, while RTN3 and Nogo-B interact with the β-secretase BACE1. Phosphorylated APP is a Pin1 substrate, which binds to its phosphor-Thr668-Pro motif. These interactions by and large resulted in a reduction of Aβ generation both in vitro and in vivo. Inhibition of Rho and Rho-kinase (ROCK) activity may underlie the ability of non-steroidal anti-inflammatory drugs and statins to reduce Aβ production, a feat which could also be achieved by Rac1 inhibition. Detailed understanding of the underlying mechanisms of action of these novel modulators of APP processing, as well as insights into the mol. neurol. basis of how Aβ impairs learning and memory, will open up multiple avenues for the therapeutic intervention of Alzheimer's disease.
CC 14-0 (Mammalian Pathological Biochemistry)
ST review amyloid precursor protein proteolytic processing Alzheimer disease
IT Alzheimer's disease
(type II; novel modulators of amyloid-β precursor protein processing)
REFERENCE COUNT: 109 THERE ARE 109 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L152 ANSWER 2 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2002:822414 HCAPLUS Full-text
DOCUMENT NUMBER: 138:131224
TITLE: The neurotrophin receptor p75NTR: novel functions and implications for diseases of the nervous system
AUTHOR(S): Dechant, Georg; Barde, Yves-Alain

CORPORATE SOURCE: Max-Planck-Institute of Neurobiology, Martinsried, 82152, Germany
SOURCE: Nature Neuroscience (2002), 5(11), 1131-1136
CODEN: NANEFN; ISSN: 1097-6256
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ED Entered STN: 29 Oct 2002
AB A review. Neurotrophins have long been known to promote the survival and differentiation of vertebrate neurons. However, these growth factors can also induce cell death through the p75 neurotrophin receptor (p75NTR), a member of the tumor necrosis factor receptor superfamily. Consistent with a function in controlling the survival and process formation of neurons, p75NTR is mainly expressed during early neuronal development. In the adult, p75NTR is re-expressed in various pathol. conditions, including epilepsy, axotomy and neurodegeneration. Potentially toxic peptides, including the amyloid β- (Aβ-) peptide that accumulates in Alzheimer's disease, are ligands for p75NTR. Recent work also implicates p75NTR in the regulation of both synaptic transmission and axonal elongation. It assocs. with the Nogo receptor, a binding protein for axonal growth inhibitors, and appears to be the transducing subunit of this receptor complex.
CC 2-0 (Mammalian Hormones)
Section cross-reference(s): 14
REFERENCE COUNT: 103 THERE ARE 103 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L152 ANSWER 3 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:873725 HCAPLUS Full-text
DOCUMENT NUMBER: 147:271226
TITLE: RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA)
INVENTOR(S): McSwiggen, James; Chowrira, Bharat M.; Haeberli, Peter
PATENT ASSIGNEE(S): Sima Therapeutics, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 195pp., Cont.-in-part of U.S. Ser. No. 826,966.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 257
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2007185043	A1	20070809	US 2007-576690	20070206 <--
AU 9851819	A	19980611	AU 1998-51819	19980112 <--
AU 729657	B2	20010208		
AU 9939188	A	19990916	AU 1999-39188	19990713 <--
AU 769175	B2	20040115	AU 2000-56616	20000911 <--
US 2005233329	A1	20051020	US 2003-727780	20031203 <--
US 2004249178	A1	20041209	US 2004-780447	20040213 <--
US 2005032733	A1	20050210	US 2004-826966	20040416 <--
WO 2005041859	A2	20050512	WO 2004-US13456	20040430 <--
WO 2005041859	A3	20070426		

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WO 2005019453	A2	20050303	WO 2004-US16390	20040524 <--
WO 2005019453	A3	20050623		

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WO 2005045035	A2	20050519	WO 2004-US26930	20040820 <--
WO 2005045035	A3	20051208		

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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2006203062	A1	20060810	AU 2006-203062	20060713 <--
AU 2006203725	A1	20060914	AU 2006-203725	20060825 <--
AU 2006228026	A1	20061102	AU 2006-228026	20061011 <--
PRIORITY APPLN. INFO.:			US 2003-693059	B2 20031023 <--
			US 2003-720448	B2 20031124 <--
			US 2003-727780	A2 20031203 <--
			US 2004-543480P	P 20040210 <--
			US 2004-780447	A2 20040213 <--
			US 2004-826966	A2 20040416 <--
			WO 2004-US13456	A 20040430 <--
			WO 2004-US16390	A 20040524 <--
			WO 2004-US26930	W 20040820 <--
			AU 1995-26422	A3 19950518 <--
			US 1996-623891	A 19960325 <--
			AU 1996-76662	A3 19961025 <--
			AU 2003-216323	A3 20030220 <--
			AU 2003-219817	A3 20030220 <--
			AU 2003-221258	A3 20030220 <--
			US 2003-427160	A2 20030430 <--
			US 2003-444853	A 20030523 <--
			US 2004-757803	A 20040114 <--

ED Entered STN: 10 Aug 2007
AB This invention relates to compds., compns., and methods useful for modulating NOGO and/or NOGO receptor gene expression using short interfering nucleic acid (siNA) mols by RNA interference. In particular, the instant invention features small nucleic acid mols., such as short interfering nucleic acid (siNA), short interfering RNA (siRNA), double-stranded RNA (dsRNA), micro-RNA (miRNA), and short hairpin RNA (shRNA) mols. and methods used to modulate the

expression of NOGO and/or NOGO receptor genes, such as NOGO-A, NOGO-B, NOGO-C, NOGO-66 receptor, NI-35, NI-220, NI-250, myelin-associated glycoprotein, tenascin-R, and NG-2. Such small nucleic acid mols. are useful in providing compns. for treatment of traits, diseases and conditions that can respond to modulation of NOGO and NOGO receptor expression in a subject for neurol. traits, diseases and conditions, such as CNS injury, cerebrovascular accident, Alzheimer's disease, dementia, multiple sclerosis, chemotherapy-induced neuropathy, macular dystrophy, amyotrophic lateral sclerosis, Parkinsons disease, ataxia, Huntington,s disease and or Creutzfeldt-Jacob disease.

INCL 514044000; 536023100

CC 6-3 (General Biochemistry)
Section cross-reference(s): 1, 3, 63

ST NOGO receptor gene expression inhibition RNA interference; short interfering nucleic acid sequence NoGO receptor; neurol disease therapy NOGO receptor siNA

IT Gene, animal
RNA
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NOGO receptor; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Diagnosis
Drug screening
Drugs
Nervous system, disease
RNA sequences
(RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Antisense nucleic acids
Double stranded RNA
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Post-transcriptional processing
(interference; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Polynucleotides
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(linker, sense region of siNA is connected to antisense region via; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT Nucleotides, biological studies
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(siNA comprises; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short

interfering nucleic acid (siNA))

IT Double stranded RNA
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(small interfering; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 50-89-5, 2'-Deoxy thymidine, biological studies 120-73-0, Purine 120-73-0D, Purine, 2'-deoxy 289-95-2, Pyrimidine 289-95-2D, Pyrimidine, 2'-Deoxy-2'-fluoro 14265-44-2, Phosphate, biological studies 55133-63-6, Methyl pyrimidine
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 56-14-4, Succinate, biological studies
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(inverted deoxyabasic, linkage; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 15181-41-6, Phosphorothioate
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(linkage, at 3'-end of antisense region, of siNA; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 69552-98-3, Glyceryl succinate
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(linkage, of siNA; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT

946028-87-1P	946028-88-2P	946028-89-3P	946028-90-6P	946028-91-7P
946028-92-8P	946028-93-9P	946028-94-0P	946028-95-1P	946028-96-2P
946028-97-3P	946028-98-4P	946028-99-5P	946029-00-1P	946029-01-2P
946029-02-3P	946029-03-4P	946029-04-5P	946029-05-6P	946029-06-7P
946029-07-8P	946029-08-9P	946029-09-0P	946029-10-3P	946029-11-4P
946029-12-5P	946029-13-6P	946029-14-7P	946029-15-8P	946029-16-9P
946029-17-0P	946029-18-1P	946029-19-2P	946029-20-5P	946029-21-6P
946029-22-7P	946029-23-8P	946029-24-9P	946029-25-0P	946029-26-1P
946029-27-2P	946029-28-3P	946029-29-4P	946029-30-7P	946029-31-8P
946029-32-9P	946029-33-0P	946029-34-1P	946029-35-2P	946029-36-3P
946029-37-4P	946029-38-5P	946029-39-6P	946029-40-9P	946029-41-0P
946029-42-1P	946029-43-2P	946029-44-3P	946029-45-4P	946029-46-5P
946029-47-6P	946029-48-7P	946029-49-8P	946029-50-1P	946029-51-2P
946029-52-3P	946029-53-4P	946029-54-5P	946029-55-6P	946029-56-7P
946029-57-8P	946029-58-9P	946029-59-0P	946029-60-3P	946029-61-4P
946029-62-5P	946029-63-6P	946029-64-7P	946029-65-8P	946029-66-9P
946029-67-0P	946029-68-1P	946029-69-2P	946029-70-5P	946029-71-6P
946029-72-7P	946029-73-8P	946029-74-9P	946029-75-0P	946029-76-1P
946029-77-2P	946029-78-3P	946029-79-4P	946029-80-7P	946029-81-8P
946029-82-9P	946029-83-0P	946029-84-1P	946029-85-2P	946029-86-3P
946029-87-4P	946029-88-5P	946029-89-6P	946029-90-9P	946029-91-0P
946029-92-1P	946029-93-2P	946029-94-3P	946029-95-4P	946029-96-5P
946029-97-6P	946029-98-7P	946029-99-8P	946030-00-8P	946030-01-9P
946030-02-0P	946030-03-1P	946030-04-2P	946030-05-3P	946030-06-4P
946030-07-5P	946030-08-6P	946030-09-7P	946030-10-0P	946030-11-1P

946030-12-2P 946030-13-3P 946030-14-4P 946030-15-5P 946030-16-6P
946030-17-7P 946030-18-8P 946030-19-9P 946030-20-2P 946030-21-3P
946030-22-4P 946030-23-5P 946030-24-6P 946030-25-7P 946030-26-8P
946030-27-9P 946030-28-0P 946030-29-1P 946030-30-4P 946030-31-5P
946030-32-6P 946030-33-7P 946030-34-8P 946030-35-9P 946030-36-0P
946030-37-1P 946030-38-2P 946030-39-3P 946030-40-6P 946030-41-7P
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946030-47-3P 946030-48-4P 946030-49-5P 946030-50-8P 946030-51-9P
946030-52-0P 946030-53-1P 946030-54-2P 946030-55-3P 946030-56-4P
946030-57-5P 946030-58-6P 946030-59-7P 946030-60-0P 946030-61-1P
946030-62-2P 946030-63-3P 946030-64-4P 946030-65-5P 946030-66-6P
946030-67-7P 946030-68-8P 946030-69-9P 946030-70-2P 946030-71-3P
946030-72-4P 946030-73-5P 946030-74-6P 946030-75-7P 946030-76-8P
946030-77-9P 946030-78-0P 946030-79-1P 946030-80-4P 946030-81-5P
946030-82-6P 946030-83-7P 946030-84-8P 946030-85-9P 946030-86-0P
946030-87-1P 946030-88-2P 946030-89-3P 946030-90-6P 946030-91-7P
946030-92-8P 946030-93-9P 946030-94-0P 946030-95-1P 946030-96-2P
946030-97-3P 946030-98-4P 946030-99-5P 946031-00-1P 946031-01-2P
946031-02-3P 946031-03-4P 946031-04-5P 946031-05-6P 946031-06-7P
946031-07-8P 946031-08-9P 946031-09-0P 946031-10-3P 946031-11-4P
946031-12-5P 946031-13-6P 946031-14-7P 946031-15-8P 946031-16-9P
946031-17-0P 946031-18-1P 946031-19-2P 946031-20-5P
RL: PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use);
BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence of short interfering nucleic acid; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 946031-21-6P 946031-22-7P 946031-23-8P 946031-24-9P 946031-25-0P
946031-26-1P 946031-27-2P 946031-28-3P 946031-29-4P 946031-30-7P
946031-31-8P 946031-32-9P 946031-33-0P 946031-34-1P 946031-35-2P
946031-36-3P 946031-37-4P 946031-38-5P 946031-39-6P 946031-40-9P
946031-41-0P 946031-42-1P 946031-43-2P 946031-44-3P 946031-45-4P
946031-46-5P 946031-47-6P 946031-48-7P 946048-81-3P 946048-82-4P
946048-83-5P 946048-84-6P 946048-85-7P 946048-86-8P 946048-87-9P
946048-88-0P 946048-89-1P 946048-90-4P 946048-91-5P 946048-92-6P
946048-93-7P 946048-94-8P 946048-95-9P 946048-96-0P 946048-97-1P
946048-98-2P 946048-99-3P 946049-00-9P 946049-01-0P 946049-02-1P
946049-03-2P 946049-04-3P 946049-05-4P 946049-06-5P 946049-08-7P
946049-10-1P 946049-11-2P 946049-12-3P 946049-13-4P 946049-14-5P
946049-15-6P 946049-16-7P 946049-17-8P 946049-18-9P 946049-19-0P
946049-20-3P 946049-21-4P 946049-22-5P 946049-23-6P 946049-24-7P
946049-25-8P 946049-27-0P 946049-33-8P 946049-37-2P 946049-38-3P
RL: PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use);
BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence of short interfering nucleic acid; RNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

IT 946032-03-7 946032-04-8 946032-05-9 946032-06-0
RL: PRP (Properties)
(unclaimed nucleotide sequence; rNA interference mediated inhibition of NOGO and NOGO receptor gene expression using short interfering nucleic acid (siNA))

L152 ANSWER 4 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:984343 HCAPLUS Full-text
TITLE: Effects of Nogo on the regeneration and diseases of central nervous system
AUTHOR(S): Yan, Ji-wen; Huang, Qi-lin

CORPORATE SOURCE: Xinqiao Hospital, Third Military Medical University, Chongqing, 400037, Peop. Rep. China
SOURCE: Chongqing Yixue (2007), 36(13), 1320-1322
CODEN: CYHIAD; ISSN: 1671-8348
PUBLISHER: Chongqing Yixue Bianjibu
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Chinese
ED Entered STN: 04 Sep 2007
AB Studies on the biol. characteristics of Nogo protein and its receptors, and the effects of Nogo on the regeneration of central nervous system (CNS) and the diseases such as Alzheimer 's disease (AD) and amyotrophic lateralizing sclerosis (ALS) are reviewed with 31 refs.
CC 14 (Mammalian Pathological Biochemistry)
ST review Nogo gene receptor central nervous system regeneration; review AD ALS
IT INDEXING IN PROGRESS
IT Alzheimer's disease
Central nervous system
Gene
Receptors
Regeneration, animal
(effects of Nogo on the regeneration and diseases of central nervous system)

L152 ANSWER 5 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:515914 HCAPLUS Full-text
DOCUMENT NUMBER: 145:1058
TITLE: Methods of cell therapy, neurogenesis and oligodendrogenesis
INVENTOR(S): Eisenbach-Schwartz, Michal; Butovsky, Oleg; Ziv, Yaniv; Kipnis, Jonathan; Ron, Noga
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel
SOURCE: PCT Int. Appl., 276 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006056998	A2	20060601	WO 2005-IL1270	20051129 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2004-631163P P 20041129 <--
ED Entered STN: 02 Jun 2006
AB A method is provided for inducing and enhancing neurogenesis and/or oligodendrogenesis from endogenous as well as from exogenously administered stem cells, which comprises administering to an individual in need a neuroprotective agent such as a nervous system (NS)-specific antigen, a

peptide derived therefrom, T cells activated therewith, poly-YE, microglia activated by interferon-γ and/or IL-4, and combinations thereof. The method includes stem cell therapy in combination with the neuroprotective agent.

IC ICM A61K
CC 1-11 (Pharmacology)
Section cross-reference(s): 15
IT Proteins
Receptors
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo, nervous system antigen; stimulating neurogenesis and oligodendrogenesis from stem cells using neuroprotective agents such as nervous system-specific antigens and poly-YE and activated T cells or microglia)
IT Alzheimer's disease
Amnesia
Anti-Alzheimer's agents
Anticonvulsants
Antigen-presenting cell
Antiglaucoma agents
Antiparkinsonian agents
Antipsychotics
Anxiety
Anxiolytics
Cell activation
Central nervous system, disease
Central nervous system agents
Cockayne's syndrome
Cognition enhancers
Cognitive disorders
Combination chemotherapy
Drug dependence
Drug withdrawal
Epilepsy
Glaucoma (disease)
Hematopoietic precursor cell
Human
Mental and behavioral disorders
Multiple sclerosis
Nervous system agents
Neurogenesis
Neuron
Oligodendrocyte
Parkinson's disease
Peripheral nervous system, disease
Schizophrenia
Sjogren syndrome
Stem cell
T cell (lymphocyte)
(stimulating neurogenesis and oligodendrogenesis from stem cells using neuroprotective agents such as nervous system-specific antigens and poly-YE and activated T cells or microglia)
IT Amyloid
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(β-, nervous system antigen; stimulating neurogenesis and oligodendrogenesis from stem cells using neuroprotective agents such as nervous system-specific antigens and poly-YE and activated T cells or microglia)
IT 888049-92-1 888049-93-2 888049-94-3 888049-95-4 888049-96-5

RL: PRP (Properties)
(unclaimed nucleotide sequence; methods of cell therapy, neurogenesis and oligodendrogenesis)
IT 888049-97-6 888049-98-7 888049-99-8 888050-00-8 888050-01-9
888050-02-0 888050-03-1 888050-04-2 888050-05-3
RL: PRP (Properties)
(unclaimed sequence; methods of cell therapy, neurogenesis and oligodendrogenesis)

L152 ANSWER 6 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:470210 HCAPLUS Full-text
DOCUMENT NUMBER: 144:482222
TITLE: Leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis
INVENTOR(S): Michalovich, David; White, Simon John; Yorke, Melanie; Maundrell, Kinsey
PATENT ASSIGNEE(S): Ares Trading S.A., Switz.
SOURCE: PCT Int. Appl., 168 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006051333	A2	20060518	WO 2005-GB4390	20051115 <--
WO 2006051333	A3	20060720		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005303536	A1	20060518	AU 2005-303536	20051115 <--
CA 2586486	A1	20060518	CA 2005-2586486	20051115 <--
EP 1814903	A2	20070808	EP 2005-803576	20051115 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				

PRIORITY APPLN. INFO.: GB 2004-25197 A 20041115 <--
WO 2005-GB4390 W 20051115

ED Entered STN: 19 May 2006
AB Four human proteins (termed INSP168, INSP168-SV1, INSP149, and INSP169) identified as leucine-rich repeat (LRR) motif containing proteins with similarity to PAL (photoreceptor-associated leucine-rich repeat protein) and to a Nogo receptor homolog are provided. The domain organization and function of these proteins allows for the design of screening methods capable of identifying compds. that are effective in the treatment and/or diagnosis of disease. INSP168 has the capacity to stimulate intracellular signaling by inducing Stat-2 nuclear translocation in the human astroglioma cell line U373, suggesting a neuroprotective role. These proteins and nucleic acid sequences

from their encoding genes are of use in the diagnosis, prevention, and treatment of disease.

CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 1, 6, 13, 63

ST leucine rich repeat protein cDNA sequence human; neuroprotection leucine rich repeat protein; disease therapy diagnosis leucine rich repeat protein

IT AIDS (disease)
(AIDS dementia complex, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(AIDS dementia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Brain, disease
Prion diseases
(Creutzfeldt-Jakob, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Nervous system, disease
(Huntington's chorea, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Protein motifs
(LRR (leucine-rich repeat); leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(Pick's disease, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Nervous system, disease
(amyotrophic lateral sclerosis, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(angioid streak, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Injury
(cerebral, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Brain, disease
(cerebrovascular, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(choroid, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(choroidal thrombosis, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(choroidal vascular insufficiency, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Brain, disease

(corticobasal degeneration, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(cystoid macular edema, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Nervous system, disease
(degeneration, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(dementia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(dementia, frontotemporal, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(dementia, vascular, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mutation
(detection of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(diabetic macular edema, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(diabetic retinopathy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Alzheimer's disease
Central nervous system, disease
Eye, neoplasm
Glaucoma (disease)
Multiple sclerosis
Parkinson's disease
Peripheral nervous system, disease
Wernicke-Korsakoff syndrome
(diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Mental and behavioral disorders
(diffuse Lewy body disease, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Gene targeting
(gene knock-out; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Eye, disease
(hereditary optic atrophy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Brain, disease
Spinal cord, disease
(injury, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy

and diagnosis)
IT Nerve, disease
(ischemia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Cardiovascular agents
Drug screening
Human
Molecular cloning
Nervous system agents
Protein sequences
Test kits
Vaccines
cDNA sequences
(leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Primers (nucleic acid)
Probes (nucleic acid)
RL: ARG (Analytical reagent use); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Antibodies and Immunoglobulins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Fusion proteins (chimeric proteins)
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(leucine-rich repeat, INSP149; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(leucine-rich repeat, INSP168-SV1; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(leucine-rich repeat, INSP168; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(leucine-rich repeat, INSP169; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(macula, senile degeneration, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

IT Diagnosis
(mol.; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Angiogenesis
(neovascularization, ocular, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Ischemia
(neuronal, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Nerve, disease
(neuropathy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Cytoprotective agents
Nervous system agents
(neuroprotective agents; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Artery, disease
Vein, disease
(occlusion, retinal, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
Inflammation
(ophthalmitis, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Nerve, disease
(optic neuropathy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Paralysis
(paraplegia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(periretinal proliferation, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(pigment epithelium, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Paralysis
(pseudobulbar, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, neoplasm
(pseudoglioma, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(retina, degeneration, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(retina, detachment, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease

therapy and diagnosis)
IT Eye, disease
(retinal ischemia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Ischemia
(retinal, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
Inflammation
(retinitis pigmentosa, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, neoplasm
(retinoblastoma, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(retinopathy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(rubeosis iritis, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
(sickle cell retinopathy, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Injury
(spinal cord, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Brain, disease
(stroke, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Brain, disease
(thalamic degeneration, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Animals
(transgenic or knockout; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Brain, disease
(trauma, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Eye, disease
Inflammation
(uveitis, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT Schizophrenia
(with dementia, diagnosis and treatment of; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT 887379-66-0P 887379-68-2P 887379-70-6P 887379-72-8P 887379-74-0P
887379-76-2P 887379-78-4P, Leucine-rich repeat (human) 887379-80-8P

887379-82-0P 887379-84-2P 887379-86-4P 887379-88-6P 887379-90-0P
887379-92-2P 887379-94-4P 887379-96-6P 887379-98-8P 887380-00-9P
887380-02-1P 887380-04-3P 887380-06-5P 887380-08-7P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT 887379-65-9P 887379-67-1P 887379-69-3P 887379-71-7P 887379-73-9P
887379-75-1P 887379-77-3P 887379-79-5P 887379-81-9P 887379-83-1P
887379-85-3P 887379-87-5P 887379-89-7P 887379-91-1P 887379-93-3P
887379-95-5P 887379-97-7P 887379-99-9P 887380-01-0P 887380-03-2P
887380-05-4P 887380-07-6P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT 887380-09-8 887380-10-1 887380-11-2 887380-12-3 887380-13-4
887380-14-5 887380-15-6 887380-16-7 887380-17-8 887380-18-9
887380-19-0 887380-20-3 887380-21-4 887380-22-5 887380-23-6
887380-24-7 887380-25-8 887380-26-9 887385-39-9 887385-40-2
RL: PRP (Properties)
(unclaimed nucleotide sequence; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)
IT 887380-27-0 887380-28-1 887380-29-2 887380-30-5
RL: PRP (Properties)
(unclaimed protein sequence; leucine-rich repeat (LRR) motif-containing proteins and their use in disease therapy and diagnosis)

L152 ANSWER 7 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:149554 HCAPLUS Full-text
DOCUMENT NUMBER: 144:226316
TITLE: Modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders
INVENTOR(S): Mi, Sha; Browning, Jeffrey L.
PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA
SOURCE: PCT Int. Appl., 138 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006017673	A2	20060216	WO 2005-US27773	20050803 <--
WO 2006017673	A3	20070412		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,

KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

CA 2576193	A1	20060216	CA 2005-2576193	20050803 <--
US 2006058223	A1	20060316	US 2005-195851	20050803 <--
EP 1789070	A2	20070530	EP 2005-783641	20050803 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
CN 101014245	A	20070808	CN 2005-80026572	20050803 <--
PRIORITY APPLN. INFO.:			US 2004-598247P P	20040803 <--
			WO 2005-US27773 W	20050803

ED Entered STN: 17 Feb 2006

AB The invention provides methods of treating diseases, disorders, injuries, or conditions involving modulating neurite outgrowth and/or survival, including central nervous system (CNS) disorders, stroke, or spinal injury, by administration of a TAJ antagonist. Purified mouse TAJ protein fused to the hinge and Fc region of human IgG1 was shown to bind the Nogo receptor 1 (NgR1) and LINGO-1 protein. Expression studies demonstrated that TAJ protein is expressed in a wide range of tissues within the mouse brain, with stronger expression during embryogenesis than during adulthood. OMGP (oligodendrocyte myelin glycoprotein) treatment of COS cells expressing TAJ, LINGO-1 and NgR1 resulted in increased RhoA activation, suggesting that expression of TAJ, LINGO-1 and NgR1 was sufficient to reconstitute a functional MAIF receptor capable of downstream signaling. Lastly, the effect of TAJ upon upon neurite outgrowth was demonstrated in rodent models. This invention is intended to be applied towards treatment of human central nervous system disorders or injuries.

CC 1-11 (Pharmacology)
Section cross-reference(s): 3, 6, 14

ST human tumor necrosis factor receptor TAJ sequence; TNFR TAJ assocn NgR1 LINGO1 signaling RhoA activation; TAJ signaling neurite outgrowth rodent model human disease therapy

IT Myelin
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(-modulated neurite outgrowth; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Nervous system, disease
(Huntington's chorea; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(LINGO-1; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo, NgR1; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Glycoproteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(OMGP (oligodendrocyte myelin glycoprotein); modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Molecular association

(TAJ association with NgR1 or LINGO-1; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Brain
(TAJ expression in; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Fusion proteins (chimeric proteins)
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(TAJ fusion with Fc IgG1; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Immunoglobulin receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(TAJ fusion with IgG1; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Tumor necrosis factor receptors
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(TAJ; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Nervous system, disease
(amyotrophic lateral sclerosis; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Epitopes
(antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Injury
(axon; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Injury
(central nervous system, transection; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Neuron
(cerebellar granule; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chimeric, antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Nerve, disease
(degeneration, CNS; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT Nerve, disease
(diabetic neuropathy; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Axon
(disease, injury; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Gene, animal
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(encoding TAJ; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Gene targeting
(gene knock-out, of TAJ gene, in mice; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(humanized, antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Central nervous system, disease
(injury, transection; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Spinal cord, disease
(injury; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Post-transcriptional processing
(interference; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Alzheimer's disease
Analgesics
Antibiotics
Astrocyte
Axon
Central nervous system
Drug screening
Genetic engineering
Human
Molecular cloning
Multiple sclerosis
Oligodendrocyte
Parkinson's disease
Signal transduction, biological
(modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Antisense nucleic acids
Corticosteroids, biological studies
Promoter (genetic element)
Rho protein (G protein)
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Molecular association

(monoclonal antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal, monoclonal antibody association with TAJ epitope; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Nerve, disease
(optic nerve injury; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Injury
(optic nerve; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Injury
(spinal cord; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Ganglion
(spinal; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Brain, disease
(stroke; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Mus musculus
(transgenic; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Brain, disease
(trauma; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT Protein sequences
(tumor necrosis factor receptor TAJ, from human; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT 876331-83-8
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT 280545-70-2, GENBANK AB040434 291801-31-5, GENBANK BAB03269
386583-22-8, GenBank AF246999 386583-24-0, GenBank AF247000
480697-79-8, GenBank AAK28396 487736-40-3, GenBank AAK28397
497742-55-9, GENBANK BC047321 623669-56-7, GENBANK AAH47321
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT 876332-29-5 876332-30-8 876332-32-0 876332-34-2 876332-36-4
876332-38-6 876332-39-7 876332-40-0 876332-41-1 876332-42-2
876332-43-3
RL: PRP (Properties)
(unclaimed nucleotide sequence; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)
IT 876332-31-9 876332-33-1 876332-35-3 876332-37-5

RL: PRP (Properties)
(unclaimed protein sequence; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

IT 122024-47-9 130838-28-7 160918-30-9 244250-73-5 278595-84-9
455901-21-0 455901-22-1 455901-23-2
RL: PRP (Properties)
(unclaimed sequence; modulation of tumor necrosis factor receptor TAJ signaling for control of neurite outgrowth in treatment of CNS disorders)

L152 ANSWER 8 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:120405 HCAPLUS Full-text
DOCUMENT NUMBER: 144:190624
TITLE: Monoclonal antibodies to human protein NOGO for the treatment and/or prophylaxis of neurological diseases
INVENTOR(S): Ellis, Jonathan Henry; Hamblin, Paul Andrew; Lewis, Alan Peter; Wilson, Paul Alexander
PATENT ASSIGNEE(S): UK
SOURCE: U.S. Pat. Appl. Publ., 95 pp., Cont.-in-part of Appl. No. PCT/GB04/05325.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006029603	A1	20060209	US 2005-177648	20050706 <--
WO 2005061544	A2	20050707	WO 2004-GB5325	20041220 <--
WO 2005061544	A3	20050818		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:			
GB 2003-29684	A	20031222 <--	
GB 2003-29711	A	20031222 <--	
WO 2004-GB5325	A2	20041220 <--	

ED Entered STN: 09 Feb 2006
AB The present invention relates to humanized antibodies to human protein NOGO, pharmaceutical formulations containing them and to the use of such antibodies in the treatment and/or prophylaxis of neurol. diseases/disorders. Provided are sequences for monoclonal humanized NOGO antibodies.
INCL 424143100; 530388220
CC 15-3 (Immunochemistry)
Section cross-reference(s): 3
IT Animal cell line
(3T3, expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Animal cell line
(CHO, expression host; monoclonal antibodies to human protein NOGO for

treatment and/or prophylaxis of neurol. diseases)
IT Animal cell line
(COS, expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Nervous system, disease
(Huntington's chorea; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Animal cell line
(NSO, expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo A, domain Nogo-A56, antibodies to; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo, antagonists; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Animal cell line
(Sp2/0, expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(chimeric; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Axon
(contacting with antibody to promote sprouting; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Nerve, disease
(degeneration, inhibiting; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Mental and behavioral disorders
(dementia, Fronto-temporal, tauopathies; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Fibroblast
(expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fragments; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(heavy chain; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized; monoclonal antibodies to human protein NOGO for

treatment and/or prophylaxis of neurol. diseases)

IT Drug delivery systems
(injections, i.v.; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Spinal cord, disease
(injury; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(light chain; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Animal cell
(mammalian, expression host; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Alzheimer's disease
Anti-Alzheimer's agents
Antiparkinsonian agents
Drug delivery systems
Human
Molecular cloning
Multiple sclerosis
Nervous system, disease
Nervous system agents
Parkinson's disease
Prophylaxis
Protein sequences
cDNA sequences
(monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Nerve, disease
(neuropathy; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(neutralizing; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Axon
(outgrowth, sprouting promotion; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Mutagenesis
(site-directed, substitution; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Injury
(spinal cord; monoclonal antibodies to human protein NOGO for

treatment and/or prophylaxis of neurol. diseases)

IT Brain, disease
(stroke; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT Brain, disease
(trauma; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 164982-65-4P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-H1; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 857289-04-4P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-H2; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 857289-05-5P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-H3; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 250143-97-6P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-L1; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 201468-24-8P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-L2; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 857289-03-3P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence, 2A10 CDR-L3; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 875356-49-3DP, mutated variants 875356-50-6DP, humanized derivs. 875356-51-7DP, humanized derivs. 875356-54-0P 875356-55-1P
875356-56-2P 875356-57-3P 875356-58-4P 875356-59-5P 875356-60-8P
875356-61-9P 875356-62-0P 875356-63-1P 875356-64-2P 875356-65-3DP, mutated variants 875356-66-4P 875356-67-5P 875356-68-6P
875356-69-7P 875356-70-0P 875356-71-1P 875356-72-2P 875356-73-3P
875356-74-4P 875356-75-5P 875356-76-6P 875356-77-7P 875356-78-8P
875356-79-9P 875356-80-2P 875356-81-3P 875356-82-4P 875356-83-5P
875356-84-6P 875356-85-7P 875356-87-9P 875356-88-0P 875356-89-1P
875356-90-4P 875356-91-5P 875356-92-6P 875356-93-7P 875356-94-8P
875356-95-9P 875356-96-0P 875356-97-1P 875356-98-2P 875356-99-3P

875357-00-9P 875357-01-0DP, mutated variants 875357-02-1P
875357-03-2P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 875356-86-8
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(amino acid sequence; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 390291-55-1, GENBANK AJ251385 392124-59-3, GenBank AJ251384
392205-86-6, GenBank AJ251383
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 875356-52-8D, derivs. 875356-53-9D, derivs.
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(nucleotide sequence; monoclonal antibodies to human protein NOGO for treatment and/or prophylaxis of neurol. diseases)

IT 875357-22-5 875357-23-6 875357-24-7 875357-25-8 875357-26-9
875357-27-0 875357-28-1 875357-29-2 875357-30-5 875357-31-6
875357-32-7 875357-33-8 875357-34-9 875357-35-0 875357-36-1
875357-37-2 875357-38-3 875357-39-4 875357-40-7 875357-41-8
875357-42-9 875357-43-0 875357-44-1 875357-45-2 875357-46-3
875357-47-4 875357-48-5 875357-49-6 875357-50-9 875357-51-0
875357-52-1 875357-53-2 875357-55-4 875357-56-5 875357-57-6
875357-58-7 875357-59-8 875357-60-1 875357-61-2 875357-62-3
875357-63-4 875357-64-5 875357-65-6 875357-66-7 875357-67-8
875357-68-9 875357-69-0 875357-70-3 875357-71-4
RL: PRP (Properties)
(unclaimed nucleotide sequence; monoclonal antibodies to human protein NOGO for the treatment and/or prophylaxis of neurol. diseases)

IT 875357-54-3 875357-72-5
RL: PRP (Properties)
(unclaimed protein sequence; monoclonal antibodies to human protein NOGO for the treatment and/or prophylaxis of neurol. diseases)

IT 247166-37-6
RL: PRP (Properties)
(unclaimed sequence; monoclonal antibodies to human protein NOGO for the treatment and/or prophylaxis of neurol. diseases)

L152 ANSWER 9 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:823596 HCAPLUS Full-text
DOCUMENT NUMBER: 143:222540
TITLE: Treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists
INVENTOR(S): Relton, Jane K.; Engber, Thomas M.; Strittmatter, Stephen M.
PATENT ASSIGNEE(S): Biogen Idec MA Inc., USA; Yale University
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005074972	A2	20050818	WO 2005-US2535	20050128 <--
WO 2005074972	A3	20051222		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005210621	A1	20050818	AU 2005-210621	20050128 <--
CA 2555018	A1	20050818	CA 2005-2555018	20050128 <--
EP 1713494	A2	20061025	EP 2005-712127	20050128 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
CN 1946418	A	20070411	CN 2005-80009242	20050128 <--
BR 2005007272	A	20070626	BR 2005-7272	20050128 <--
JP 2007519737	T	20070719	JP 2006-551456	20050128 <--
MX 2006PA08392	A	20061030	MX 2006-PA8392	20060725 <--
IN 2006DN04365	A	20070831	IN 2006-DN4365	20060728 <--
KR 2007052237	A	20070521	KR 2006-717342	20060828 <--
PRIORITY APPLN. INFO.:			US 2004-540798P P	20040130 <--
			WO 2005-US2535 W	20050128

ED Entered STN: 19 Aug 2005

AB The invention provides methods for promoting regeneration or survival of dopaminergic neurons in a mammal displaying signs or symptoms of dopaminergic neuronal degeneration, including a human with Parkinson's disease, using Nogo receptor antagonists. The number of surviving dopaminergic neurons in the substantia nigra was significantly greater in Nogo receptor knockout mice compared to their heterozygote and wild-type litter-mate controls 4 wk after unilateral 6-hydroxydopamine injections. In addition, rotational behavior in response to apomorphine challenge was significantly lower in Nogo receptor-null mice. These data show increased neuronal survival and improved recovery of function in dopaminergic pathways in the brain after injury in mice lacking Nogo receptor. Treatment with the Nogo receptor antagonist sNgR(310)-Fc (soluble mature Nogo receptor fused with an Ig Fc fragment) increases cell survival and improved recovery in dopaminergic pathways in rat brain after injury. Thus, Nogo receptor antagonists comprising soluble Nogo receptor polypeptides, antibodies to the Nogo receptor protein, and small mol. may promote regeneration and survival of dopaminergic neurons in mammals displaying degeneration.

IC ICM A61K038-17
ICS C07K016-28; A61P025-00; A61P025-28

CC 1-11 (Pharmacology)

ST dopaminergic neuron degeneration Nogo receptor antagonist

IT Nervous system, disease
(Hallervorden-Spatz disease; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease

(Huntington's chorea; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease
(Machado-Joseph; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Receptors
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Disease, animal
(Shy-Drager syndrome; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease
(X-linked dystonia-parkinsonism; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Movement disorders
(cerebral palsy; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Brain
(corpus striatum; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease
(degeneration; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(diabodies; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Mental and behavioral disorders
(diffuse Lewy body disease; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Phagocyte
(disease, Chediak-Higashi syndrome; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nerve
(dopaminergic, disease, degeneration; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fragments; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(fusion products; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nerve, disease
(motor; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease
(multiple system atrophy; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Syphilis
(neuro-; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Parkinson's disease
(postencephalitic; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Paralysis
(pseudobulbar; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(single chain; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Nervous system, disease
(spinocerebellar ataxia; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Brain
(striatonigral tract, disease, degeneration; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Brain
(substantia nigra; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Head and Neck, disease
(trauma; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Alzheimer's disease
Antiparkinsonian agents
Human
Nerve regeneration
Nerve regeneration
Nervous system agents
Parkinson's disease
Prion diseases
Protein sequences

Rattus
Wilson's disease
(treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Antibodies and Immunoglobulins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT Parkinson's disease
(vascular; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT 862861-59-4, Nogo receptor (human precursor)
862861-60-7, Nogo receptor (Rattus precursor)
862861-61-8, 1-310-Nogo receptor (human)
862861-62-9, Nogo receptor (human) 862861-63-0, 1-310-Nogo receptor (Rattus) 862861-64-1, Nogo receptor (Rattus)
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

IT 783350-09-4 783350-10-7 783350-11-8
783350-12-9 783350-13-0 783350-14-1
783350-15-2 783350-16-3 783350-17-4
783350-18-5 783350-19-6 783350-20-9
783350-21-0 783350-22-1 783350-23-2
783350-24-3
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(peptide fragment of Nogo receptor; treatment of conditions involving dopaminergic neuronal degeneration using Nogo receptor antagonists)

L152 ANSWER 10 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:589035 HCAPLUS Full-text
DOCUMENT NUMBER: 143:114054
TITLE: Humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease
INVENTOR(S): Hussain, Ishrut; Prinjha, Rabinder Kumar
PATENT ASSIGNEE(S): Glaxo Group Limited, UK
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005061545	A2	20050707	WO 2004-GB5343	20041220 <--
WO 2005061545	A3	20050818		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1706426 A2 20061004 EP 2004-806145 20041220 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, HR, IS

PRIORITY APPLN. INFO.: GB 2003-29684 A 20031222 <--
GB 2003-29711 A 20031222 <--
WO 2004-GB5343 W 20041220 <--

ED Entered STN: 08 Jul 2005

AB Methods of modulating production of an amyloidogenic peptide is disclosed. Use of such methods in the treatment of diseases involving amyloidosis, for example Alzheimer's disease, is also disclosed.

IC ICM C07K016-22
ICS A61P025-28; C12N005-08

CC 15-3 (Immunochemistry)

ST human Nogo NogoA protein humanized antibody amyloidosis neurodegenerative disease; Alzheimer disease amyloidogenic peptide modulator antibody Nogo antagonist

IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo, antagonist; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NogoA antagonist; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Peptides, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(amyloidogenic; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Nervous system, disease
(degeneration; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fragments; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(heavy chain; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Alzheimer's disease
Amyloidosis
DNA sequences
Human
Molecular cloning
Protein sequences
(humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Amyloid precursor proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(light chain; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT Amyloid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β-; humanized anti-human Nogo or NogoA protein antibodies and antagonists for

treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT 857329-28-3DP, humanized 857329-91-0DP, humanized 857329-92-1DP, humanized 857329-93-2DP, humanized 857329-94-3DP, humanized 857329-95-4DP, humanized
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT 149970-72-9P 151145-43-6P 158329-15-8P 164982-65-4P 201468-24-8P
209329-62-4P 250143-97-6P 263365-34-0P 380538-12-5P 380538-13-6P
590368-20-0P 857289-03-3P 857289-04-4P 857289-05-5P 857289-06-6P
857289-07-7P 857289-08-8P 857289-09-9P 857329-29-4P 857329-30-7P
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857329-36-3P 857329-37-4P 857329-38-5P 857329-39-6P 857329-40-9P
857329-41-0P 857329-42-1P 857329-43-2P 857329-44-3P 857329-45-4P
857329-46-5P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT 857329-90-9DP, humanized 857329-96-5DP, humanized 857329-97-6DP, humanized 857329-98-7DP, humanized 857329-99-8DP, humanized 857330-00-8DP, humanized 857330-01-9DP, humanized 857330-02-0DP, humanized
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

IT 857330-03-1 857330-04-2 857330-05-3 857330-06-4 857330-07-5
857330-08-6 857330-09-7 857330-10-0 857330-11-1 857330-12-2
857330-13-3 857330-14-4 857330-15-5 857330-16-6 857330-17-7
857330-18-8 857330-19-9 857330-20-2 857330-21-3 857330-22-4
857330-23-5 857330-24-6 857330-25-7 857330-26-8 857330-27-9
857330-28-0 857330-29-1 857330-30-4 857330-31-5 857330-32-6
857330-33-7
RL: PRP (Properties)
(unclaimed nucleotide sequence; humanized anti-human Nogo or NogoA protein antibodies and antagonists for treating neurodegenerative disease, amyloidosis and Alzheimer's disease)

L152 ANSWER 11 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:589033 HCAPLUS Full-text
DOCUMENT NUMBER: 143:114053
TITLE: Human NOGO protein-specific antibodies and derivatives for treatment of stroke or other neurological diseases
INVENTOR(S): Ellis, Jonathan Henry; Eon-Duval, Alexandre; Grundy, Robert Ian; Hussain, Farhana; McAdam, Ruth; Plumpton, Christopher; Prinjha, Rabinder Kumar; Wilson, Paul

Alexander
PATENT ASSIGNEE(S): Glaxo Group Limited, UK
SOURCE: PCT Int. Appl., 143 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005061544	A2	20050707	WO 2004-GB5325	20041220 <--
WO 2005061544	A3	20050818		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004303585	A1	20050707	AU 2004-303585	20041220 <--
CA 2549956	A1	20050707	CA 2004-2549956	20041220 <--
EP 1711530	A2	20061018	EP 2004-806128	20041220 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, HR, IS				
CN 1918180	A	20070221	CN 2004-80041952	20041220 <--
BR 2004017959	A	20070327	BR 2004-17959	20041220 <--
US 2006029603	A1	20060209	US 2005-177648	20050706 <--
IN 2006KN01589	A	20070504	IN 2006-KN1589	20060608 <--
NO 2006002767	A	20060920	NO 2006-2767	20060613 <--
US 2007065430	A1	20070322	US 2006-583877	20060621 <--
MX 2006PA07190	A	20060823	MX 2006-PA7190	20060622 <--

PRIORITY APPLN. INFO.:
GB 2003-29684 A 20031222 <--
GB 2003-29711 A 20031222 <--
WO 2004-GB5325 W 20041220 <--

ED Entered STN: 08 Jul 2005
AB The present invention relates to antibodies to NOGO, pharmaceutical formulations containing them and to the use of such antibodies in the treatment and/or prophylaxis of neurol. diseases/disorders.
IC ICM C07K016-22
ICS A61P025-28; A61P025-00
CC 15-3 (Immunochemistry)
Section cross-reference(s): 3, 63
IT Animal cell line
(3T3; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Animal cell line
(CHO; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Animal cell line
(COS; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Nervous system, disease
(Huntington's chorea; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Proteins

RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOGO-A; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOGO-B; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NOGO-C; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Fusion proteins (chimeric proteins)
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOGO; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Animal cell line
(NSO; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Animal cell line
(Sp2/0; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Drug delivery systems
(carriers; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Injury
(cerebral; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(chimeric; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Nervous system, disease
(degeneration; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Mental and behavioral disorders
(dementia, fronto-temporal; tauopathy; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fragments; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)
IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified);

PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(heavy chain; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Alzheimer's disease
Animal tissue culture
Culture media
DNA sequences
Dissociation constant
Drug delivery systems
Fibroblast
Genetic vectors
Human
Molecular cloning
Multiple sclerosis
Nervous system, disease
Parkinson's disease
Protein sequences
(human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Drug delivery systems
(injections, i.v.; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Brain, disease
Spinal cord, disease
(injury; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(light chain; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Animal cell
(mammalian; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal, neutralizing; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Nerve, disease
(neuropathy; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Antibodies and Immunoglobulins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(neutralizing; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Axon
(outgrowth, sprouting promotion; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Injury
(spinal cord; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Brain, disease
(stroke; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT Brain, disease
(trauma; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT 857508-25-9DP, humanized or chimeric derivs. 857512-40-4DP, humanized or chimeric derivs. 857512-41-5DP, humanized or chimeric derivs. 857512-42-6DP, humanized or chimeric derivs. 857512-43-7DP, humanized or chimeric derivs. 857512-44-8DP, humanized or chimeric derivs. 857512-53-9DP, humanized or chimeric derivs. 857512-54-0DP, humanized or chimeric derivs. 857512-55-1DP, humanized or chimeric derivs. 857512-56-2DP, humanized or chimeric derivs. 857512-57-3DP, Protein NOGO-A56 (human), humanized or chimeric derivs. 857512-58-4DP, humanized or chimeric derivs. 857512-59-5DP, humanized or chimeric derivs. 857512-62-0DP, humanized or chimeric derivs. 857512-64-2DP, humanized or chimeric derivs.
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT 149970-72-9P 151145-43-6P 158329-15-8P 164982-65-4P 201468-24-8P 209329-62-4P 250143-97-6P 263365-34-0P 380538-12-5P 380538-13-6P 590368-20-0P 857289-03-3P 857289-04-4P 857289-05-5P 857289-06-6P 857289-07-7P 857289-08-8P 857289-09-9P 857508-26-0P 857508-27-1P 857508-28-2P 857508-29-3P 857508-30-6P 857508-31-7P 857508-32-8P 857508-33-9P 857508-34-0P 857508-35-1P 857508-36-2P 857508-37-3P 857508-38-4P 857508-39-5P 857508-40-8P 857508-41-9P 857508-42-0P 857508-43-1P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT 195861-53-1D, GenBank U84162, chimeric derivs. 390291-55-1, GenBank AJ251385 392124-59-3, GenBank AJ251384 392205-86-6, GenBank AJ251383 480670-48-2D, GenBank CAA85593, chimeric derivs.
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT 857512-45-9DP, humanized or chimeric derivs. 857512-46-0DP, humanized or chimeric derivs. 857512-47-1DP, humanized or chimeric derivs.

857512-48-2DP, humanized or chimeric derivs. 857512-49-3DP, humanized or chimeric derivs. 857512-50-6DP, humanized or chimeric derivs. 857512-51-7DP, humanized or chimeric derivs. 857512-52-8DP, humanized or chimeric derivs. 857512-60-8DP, humanized or chimeric derivs. 857512-61-9DP, humanized or chimeric derivs. 857512-63-1DP, humanized or chimeric derivs. 857512-65-3DP, humanized or chimeric derivs.
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

IT 247166-37-6 857514-52-4 857514-53-5 857514-54-6 857514-55-7
857514-56-8 857514-57-9 857514-58-0 857514-59-1 857514-60-4
857514-61-5 857514-62-6 857514-63-7 857514-64-8 857514-65-9
857514-66-0 857514-67-1 857514-68-2 857514-69-3 857514-70-6
857514-71-7 857514-72-8 857514-73-9 857514-74-0 857514-75-1
857514-76-2 857514-77-3 857514-78-4 857514-79-5 857514-80-8
857514-81-9 857514-82-0
RL: PRP (Properties)
(unclaimed sequence; human NOGO protein-specific antibodies and derivs. for treatment of stroke or other neurol. diseases)

L152 ANSWER 12 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:564795 HCAPLUS Full-text
DOCUMENT NUMBER: 143:91068
TITLE: Methods of stimulating axonal growth of CNS neurons using Nogo receptor antagonists in combination with growth factors
INVENTOR(S): Benowitz, Larry I.; Fischer, Dietmar
PATENT ASSIGNEE(S): Children's Medical Center Corporation, USA
SOURCE: PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005059515	A2	20050630	WO 2004-US42255	20041216 <--
WO 2005059515	A3	20060908		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2549000	A1	20050630	CA 2004-2549000	20041216 <--
EP 1695061	A2	20060830	EP 2004-814439	20041216 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
JP 2007514748	T	20070607	JP 2006-545428	20041216 <--
PRIORITY APPLN. INFO.:			US 2003-529833P P	20031216 <--
			WO 2004-US42255 W	20041216 <--

ED Entered STN: 30 Jun 2005
AB The invention is based on the discovery that suppressing the activity of the Nogo receptor (NgR) alone does not result in extensive axon regeneration unless the intrinsic growth program of neurons is also activated. Accordingly, the invention is directed to methods of stimulating axon regeneration using a combination therapy wherein agents that inhibit NgR activity or downstream pathways activated by inhibitory signals are combined with agents that activate the growth pathway of neurons (e.g. polypeptide growth factors, activators of macrophages, purine nucleosides, or hexoses). The invention provides protein sequences for Nogo receptor peptide antagonists, including soluble Nogo receptor fragments. Rats were injected with an adeno-associated viral vector expressing Nogo receptor or a dominant-neg. Nogo receptor (NgRDN). After nerve crush and lens injury, animals expressing NgRDN had approx. 3x more axon extensions than control animals expressing GFP reporter and 75x more axon extensions than animals expressing wild-type NgR. In another example, RhoA protein was inactivated by transgenic expression of Clostridium botulinum C3 ADP-ribosyltransferase. After lens injury, to activate the growth state of retinal ganglion cells, animals expressing the C3 transgene had 4.5x more axons that extended far beyond the injury site compared with uninjured animals expressing C3 transgene or non-transgenic injured animals. In both examples, the effects of transgenes on retinal ganglion cell growth were greater when cells were grown on myelin, an inhibitory substrate.
IC ICM G01N
CC 1-11 (Pharmacology)
Section cross-reference(s): 2, 3, 6, 13, 15
ST CNS neuron regeneration method Nogo receptor antagonist growth factor; protein sequence Nogo receptor peptide antagonist
IT Growth factors, animal
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(CNTF; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Nervous system, disease
(Friedreich's ataxia; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Brain, disease
(Gilles de la Tourette syndrome; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Nervous system, disease
(Huntington's chorea; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Growth factors, animal
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NT-3; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Antibodies and Immunoglobulins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NgR antagonist or NgR ligand-binding; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)
IT Peptides, biological studies
RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(NgR antagonists; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Receptors
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(Nogo; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Mental and behavioral disorders
(Pick's disease; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Rho protein (G protein)
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(RhoA, NgR signaling; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Disease, animal
(Shy-Drager syndrome; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nervous system, disease
(amyotrophic lateral sclerosis; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Brain, disease
(aneurysm; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Disease, animal
(atrophy, diffuse cerebral cortical; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Muscle, disease
(atrophy; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Neurotrophic factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(brain-derived; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT G protein-coupled receptors
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(cAMP signaling; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Ionophores
(calcium, cAMP signaling; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Aneurysm

(cerebral; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nervous system, disease
(chorea, acanthocytic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Eye, disease
(chronic progressive external ophthalmoplegia; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nervous system, disease
(degeneration, Hallervorden-Spatz disease; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Brain, disease
(degeneration, thalamic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Mental and behavioral disorders
(dementia, Gerstmann-Straussler-Scheinker disease; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Mental and behavioral disorders
(dementia, mesolimbocortical; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Mental and behavioral disorders
(diffuse Lewy body disease; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Mutagenesis
(dominant-neg. NgR; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nervous system, disease
(dyskinesia, Meige syndrome; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nervous system, disease
(dystonia musculorum deformans; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Tremor
(familial; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Transgene
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(for ADP-ribosyltransferase; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Gene, animal
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(for NgR; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Neurotrophic factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(glial-derived; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(injections, s.c.; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Spinal cord, disease
(injury; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(intrathecal; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(intraventricular; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Adeno-associated virus
Adeno-associated virus 2
Alzheimer's disease
Aptamers
Axon
Central nervous system
Gene therapy
Human
Mammalia
Nerve
Nervous system agents
Parkinson's disease
Protein sequences
Rattus
Regeneration, animal
Signal transduction, biological
Spinal column, disease
Spinal muscular atrophy
Viral vectors
(methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Hexoses
Interleukin 6
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(nasal; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nerve, disease
(neuropathy, optic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Proteins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oncomodulins; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(ophthalmic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(oral; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Paralysis
(paraparesis, tropical spastic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Paralysis
(paraplegia, spastic; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(parenterals; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Nerve, disease
(polyneuropathy; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Paralysis
(progressive balbar; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Paralysis
(pseudobulbar; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Eye
(retina; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Injury
(spinal cord; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Brain, disease
(stroke; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Drug delivery systems
(topical; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Brain, disease
(trauma; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(β-; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 58319-92-9P, ADP-ribosyltransferase
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(Clostridium botulinum C3; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 350811-10-8P 350811-11-9P 350811-12-0P 856266-29-0P
RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(Nogo receptor antagonist peptide ; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 856266-30-3P 856266-38-1P 856266-39-2P 856266-40-5P, 1-344-Nogo receptor (human) 856266-41-6P, 1-310-Nogo receptor (human) 856266-42-7P, 1-344-Nogo receptor (Rattus) 856266-43-8P, 1-310-Nogo receptor (Rattus) 856266-45-0P 856266-46-1P, 26-344-Nogo receptor (human) 856266-47-2P, 26-310-Nogo receptor (human) 856266-48-3P, 26-344-Nogo receptor (Rattus) 856266-49-4P, 27-344-Nogo receptor (Rattus) 856266-50-7P, 27-310-Nogo receptor (Rattus)
RL: BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 856266-44-9D, 1055-1120-Protein Nogo A (human), N-terminal amidated, C-terminal acylated
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 7440-70-2, Calcium, biological studies 9012-42-4, Adenylate cyclase
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(cAMP signaling; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 9036-21-9, CAMP phosphodiesterase 9040-59-9, 3',5'-Cyclic nucleotide phosphodiesterase
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(inhibitors, cAMP signaling; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 60-92-4, Cyclic AMP
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(intracellular; methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 508116-22-1, GENBANK AF532858
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(methods of stimulating axonal growth of CNS neurons using Nogo receptor (NgR) antagonists in combination with growth factors)

IT 56-73-5, Glucose-6-phosphate 58-63-9, Inosine 3458-28-4, D-Mannose 9061-61-4, Nerve growth factor 19163-87-2, Gulose
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods of stimulating axonal growth of CNS neurons using Nogo

receptor (NgR) antagonists in combination with growth factors)

IT 856273-14-8 856273-15-9
RL: PRP (Properties)
(unclaimed nucleotide sequence; methods of stimulating axonal growth of CNS neurons using Nogo receptor antagonists in combination with growth factors)

L152 ANSWER 13 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:266024 HCAPLUS Full-text
DOCUMENT NUMBER: 144:387731
TITLE: Reticulons and β-secretase
AUTHOR(S): Araki, Wataru
CORPORATE SOURCE: Dep. Demyelinating Dis. Aging, Natl. Inst. Neurosci., National Center of Neurology and Psychiatry (NCNP), Kodaira, 187-8502, Japan
SOURCE: Dementia Japan (2005), 19(3), 266-272
CODEN: DEJAFB; ISSN: 1342-646X
PUBLISHER: Nippon Chiho Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
ED Entered STN: 23 Mar 2006

AB A review. Cerebral accumulation of amyloid β- protein (Aβ) is the main pathol. feature of Alzheimer's disease (AD). β-Secretase cleavage of amyloid precursor protein (APP) is catalyzed by the membrane-bound aspartyl protease BACE (β-site APP cleaving enzyme). Inhibition of BACE is one of the attractive therapeutic approaches for AD. Recently, we and others identified Nogo-B (reticulon 4-B) and its homolog reticulon 3 as BACE-interacting membrane proteins. These reticulon family proteins appear to neg. modulate Aβ production through phys. association with BACE. The role of reticulon proteins in the regulation of BACE function is discussed.

CC 14-0 (Mammalian Pathological Biochemistry)
ST review reticulon secretase BACE Alzheimer disease
IT Proteins
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(Nogo; reticulons and β-secretase (BACE) in amyloid β-protein accumulation in Alzheimer's disease)

IT Proteins
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(membrane, BACE-interacting, reticulon 3; reticulons and β-secretase (BACE) in amyloid β-protein accumulation in Alzheimer's disease)

IT Alzheimer's disease
Brain
Drug targets
(reticulons and β-secretase (BACE) in amyloid β-protein accumulation in Alzheimer's disease)

IT Amyloid precursor proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(reticulons and β-secretase (BACE) in amyloid β-protein accumulation in Alzheimer's disease)

IT Amyloid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β-; reticulons and β-secretase (BACE) in amyloid

β-protein accumulation in Alzheimer's disease)

IT 158736-49-3, β-Site APP cleaving enzyme
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(reticulons and β-secretase (BACE) in amyloid β-protein accumulation in Alzheimer's disease)

L152 ANSWER 14 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:927061 HCAPLUS Full-text
DOCUMENT NUMBER: 141:406109
TITLE: Treatment of conditions involving amyloid plaques
INVENTOR(S): Strittmatter, Stephen M.; Lee, Daniel H. S.; Li, Weiwei
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004093893	A2	20041104	WO 2004-US11728	20040416 <--
WO 2004093893	A3	20050303		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004231742	A2	20041104	AU 2004-231742	20040416 <--
AU 2004231742	A1	20041104		
CA 2522649	A1	20041104	CA 2004-2522649	20040416 <--
EP 1615654	A2	20060118	EP 2004-759905	20040416 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
BR 2004009562	A	20060418	BR 2004-9562	20040416 <--
CN 1832752	A	20060913	CN 2004-80016919	20040416 <--
JP 2006523708	T	20061019	JP 2006-510107	20040416 <--
MX 2005PA11100	A	20060418	MX 2005-PA11100	20051014 <--
IN 2005DN04897	A	20070817	IN 2005-DN4897	20051026 <--
NO 2005005392	A	20051115	NO 2005-5392	20051115 <--
US 2007065429	A1	20070322	US 2006-553669	20060809 <--
PRIORITY APPLN. INFO.:			US 2003-463424P P	20030416 <--
			WO 2004-US11728 W	20040416 <--

ED Entered STN: 04 Nov 2004
AB The invention provides methods for treating diseases involving aberrant amyloid-β (Aβ) peptide deposition, including Alzheimer's Disease, by the administration of Nogo receptor antagonists. The invention also provides method for reducing levels of Aβ peptide in a mammal by the administration of soluble Nogo receptor polypeptides.

IC ICM A61K038-00
CC 1-11 (Pharmacology)
Section cross-reference(s): 3
ST amyloid plaque Alzheimer disease polypeptide
IT Amyloid precursor proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(APP695; treatment of conditions involving amyloid plaques)
IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; treatment of conditions involving amyloid plaques)
IT Drug delivery systems
(infusions; treatment of conditions involving amyloid plaques)
IT Drug delivery systems
(injections; treatment of conditions involving amyloid plaques)
IT Brain
(lateral ventricle; treatment of conditions involving amyloid plaques)
IT Alzheimer's disease
Anti-Alzheimer's agents
Central nervous system
Human
(treatment of conditions involving amyloid plaques)
IT Signal peptides
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(treatment of conditions involving amyloid plaques)
IT Amyloid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β-; treatment of conditions involving amyloid plaques)
IT 786653-00-7, HB 7E11 786653-17-6, HB 1H2
786653-18-7, HB 3G5 786653-21-2, HB 5B10
786653-25-6, HB 2F7
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(treatment of conditions involving amyloid plaques)
IT 783350-09-4 783350-10-7 783350-11-8
783350-12-9 783350-13-0, LDLASDDAELR 783350-14-1
, LDALSDNAQLR 783350-15-2, LDALSDDAELR 783350-16-3,
LDLSSDNAQLR 783350-17-4, LDLSSDEAELR 783350-18-5,
DNAQLRVVDPTT 783350-19-6, DNAQLR 783350-20-9,
ADLSDNAQLRVVDPTT 783350-21-0, LALSDNAQLRVVDPTT
783350-22-1, LDLSDNAALRVVDPTT 783350-23-2,
LDLSDNAQLHVVDPTT 783350-24-3, LDLSDNAQLAVVDPTT
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(treatment of conditions involving amyloid plaques)
IT 790777-25-2 790777-26-3 790777-27-4
790777-28-5 790777-29-6 790777-30-9
RL: PRP (Properties)
(unclaimed protein sequence; treatment of conditions involving amyloid plaques)

L152 ANSWER 15 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:824033 HCAPLUS Full-text
DOCUMENT NUMBER: 141:290091
TITLE: Protein and cDNA sequences of a novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for

neuron diseases

INVENTOR(S): Mi, Sha; McCoy, John; Pepinsky, R. Blake; Lee, Daniel H. S.

PATENT ASSIGNEE(S): Biogen Idec Ma Inc., USA

SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085648	A2	20041007	WO 2004-US8323	20040317 <--
WO 2004085648	A3	20041118		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004223464	A2	20041007	AU 2004-223464	20040317 <--
AU 2004223464	A1	20041007		
CA 2519227	A1	20041007	CA 2004-2519227	20040317 <--
EP 1606409	A2	20051221	EP 2004-757823	20040317 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
BR 2004008501	A	20060314	BR 2004-8501	20040317 <--
CN 1798840	A	20060705	CN 2004-80013836	20040317 <--
JP 2007524370	T	20070830	JP 2006-507330	20040317 <--
IN 2005DN04139	A	20070831	IN 2005-DN4139	20050914 <--
US 2007059793	A1	20070315	US 2005-553685	20051017 <--
NO 2005004836	A	20051019	NO 2005-4836	20051019 <--

PRIORITY APPLN. INFO.:			
US 2003-455756P	P	20030319	<--
US 2003-480241P	P	20030620	<--
US 2003-492057P	P	20030801	<--
WO 2004-US8323	W	20040317	<--

ED Entered STN: 08 Oct 2004

AB The invention provides Sp35 polypeptides and fusion proteins thereof, Sp35 antibodies and antigen-binding fragments thereof and nucleic acids encoding the same. The invention also provides compns. comprising, and methods for making and using, such Sp35 antibodies, antigen-binding fragments thereof, Sp35 polypeptides and fusion proteins thereof.

IC ICM C12N015-12
ICS C12N015-62; C12N015-63; C07K014-47; C07K016-18; A61K038-17; A61K039-395; A61K048-00

CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 1, 6, 13

ST protein sequence human Nogo receptor binding Sp35 neuron disease

IT Nervous system, disease
(Huntington's chorea, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Proteins
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(LINGO, Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Signal transduction, biological
(NgR1, inhibition of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Fusion proteins (chimeric proteins)
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Gene, animal
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Nervous system, disease
(amyotrophic lateral sclerosis, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Carbohydrates, biological studies
Polyoxyalkylenes, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(conjugated with protein Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Polymers, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(conjugates, with protein Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Nervous system, disease
(degeneration, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Nerve, disease
(diabetic neuropathy, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(fragments, Fc, fused with Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

IT Drug delivery systems
(injections, s.c.; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35

and therapeutic use for neuron diseases)
IT Spinal cord, disease
(injury, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Drug delivery systems
(intrathecal, subdural; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Repeat motifs (protein)
(leucine-rich repeat; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Drug delivery systems
(ophthalmic; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Nervè, disease
(optic nerve injury, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Injury
(optic nerve, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Axon
(outgrowth; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Drug delivery systems
(parenterals; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Adenoviral vectors
Anti-Alzheimer's agents
Antiparkinsonian agents
Baculoviridae
Central nervous system
Gene therapy
Human
Human herpesvirus
Human herpesvirus 4
Lentiviral vectors
Mammalia
Molecular cloning
Nervous system agents
Protein sequences
Vaccinia virus
Viral vectors
cDNA sequences
(protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Albumins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(serum, fused with Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Injury
(spinal cord, treatment of; protein and cDNA sequences of

novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Brain, disease
(stroke, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(to Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Drug delivery systems
(topical; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Brain, disease
(trauma, treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT Alzheimer's disease
Central nervous system, disease
Multiple sclerosis
Parkinson's disease
(treatment of; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT 762413-55-8DP, Protein Sp35 (human), subfragments are claimed
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(amino acid sequence; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT 25322-68-3, Peg
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(conjugated with protein Sp35; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT 762413-54-7
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; protein and cDNA sequences of novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT 762413-63-8 762413-64-9 762413-65-0 762413-66-1 762413-67-2
762413-68-3 762413-69-4 762413-70-7 762413-71-8 762413-72-9
762413-73-0 762413-74-1 762413-75-2 762413-76-3 762413-77-4
762413-78-5 762413-79-6 762413-80-9 762413-81-0 762413-82-1
762413-83-2 762413-84-3 762413-85-4 762413-86-5 762413-87-6
762413-88-7 762413-89-8
RL: PRP (Properties)
(unclaimed nucleotide sequence; protein and cDNA sequences of a novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)
IT 762273-66-5 762273-67-6 762273-68-7 762273-69-8 762273-70-1
762273-71-2 762273-72-3 762273-73-4 762273-74-5 762273-75-6
762273-76-7
RL: PRP (Properties)
(unclaimed sequence; protein and cDNA sequences of a novel human Nogo receptor 1 (NgR1) binding protein Sp35 and therapeutic use for neuron diseases)

L152 ANSWER 16 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:142908 HCAPLUS Full-text
DOCUMENT NUMBER: 140:198086
TITLE: Nogo receptor antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury
INVENTOR(S): Lee, Daniel H. S.; Pepinsky, R. Blake; Li, Weiwei; Rabacchi, Sylvia A.; Relton, Jane K.; Worley, Dane S.; Strittmatter, Stephen M.; Sah, Dinah Y. W.
PATENT ASSIGNEE(S): Yale University, USA; Biogen, Inc.
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004014311	A2	20040219	WO 2003-US25004	20030807 <--
WO 2004014311	A3	20040429		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2495121	A1	20040219	CA 2003-2495121	20030807 <--
AU 2003264033	A1	20040225	AU 2003-264033	20030807 <--
EP 1534736	A2	20050601	EP 2003-785123	20030807 <--
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CN 1681838	A	20051012	CN 2003-821409	20030807 <--
JP 2005535329	T	20051124	JP 2004-527960	20030807 <--
BR 2003013331	A	20070724	BR 2003-13331	20030807 <--
AU 2004264405	A1	20050224	AU 2004-264405	20040130 <--
CA 2535007	A1	20050224	CA 2004-2535007	20040130 <--
WO 2005016955	A2	20050224	WO 2004-US2702	20040130 <--
WO 2005016955	A3	20060720		
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RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1660517	A2	20060531	EP 2004-707073	20040130 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2004013426	A	20061017	BR 2004-13426	20040130 <--
JP 2007501612	T	20070201	JP 2006-522535	20040130 <--

CN 1926147	A	20070307	CN 2004-80029412	20040130 <--
NO 2005000685	A	20050510	NO 2005-685	20050209 <--
MX 2005PA01615	A	20050819	MX 2005-PA1615	20050210 <--
US 2005271655	A1	20051208	US 2005-55163	20050210 <--
IN 2005KN00382	A	20060512	IN 2005-KN382	20050309 <--
MX 2006PA01444	A	20060515	MX 2006-PA1444	20060203 <--
NO 2006001081	A	20060418	NO 2006-1081	20060306 <--
IN 2006DN01161	A	20070810	IN 2006-DN1161	20060306 <--

PRIORITY APPLN. INFO.:

US 2002-402866P	P	20020810 <--
WO 2003-US25004	W	20030807 <--
WO 2003-US325004	A	20030807 <--
WO 2004-US2702	W	20040130 <--

ED Entered STN: 22 Feb 2004

AB Disclosed are immunogenic Nogo receptor-1 polypeptides, Nogo receptor-1 antibodies, antigen-binding fragments thereof, soluble Nogo receptors and fusion proteins thereof and nucleic acids encoding the same. Also disclosed are compns. comprising, and methods for making and using, such Nogo receptor antibodies, antigen-binding fragments, humanized and chimeric antibodies thereof, soluble Nogo receptors and fusion proteins thereof and nucleic acids or viral vector encoding the same for gene therapy. These Nogo receptor-1, antagonists are useful for inhibiting growth cone collapse of neuron, decreasing inhibition of neurite outgrowth, promoting survival of CNS neuron and axonal growth, and are therefore useful for treating multiple sclerosis, ALS, Huntington's disease, Alzheimer's disease, Parkinson's disease, diabetes neuropathy, stroke, traumatic brain injury or spinal cord injury.

IC ICM A61K
CC 15-3 (Immunochemistry)
Section cross-reference(s): 1, 63
ST Nogo receptor antibody fragment soluble fusion protein survival neuron; axonal growth Nogo receptor antagonist gene humanized chimeric antibody
IT Protein motifs
(C-terminal LRR domain; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)
IT Nervous system, disease
(Huntington's chorea; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(IgG; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)
IT Protein motifs
(LRR; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)
IT Glycoproteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MAG (myelin-associated glycoprotein); Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)
IT Protein motifs
(N-terminal domain; Nogo receptor-1

antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Alzheimer's disease
Animal cell line
Animals
Central nervous system, disease
Culture media
Drugs
Gene therapy
Genetic vectors
Human
Mammalia
Molecular cloning
Multiple sclerosis
Mus
Parkinson's disease
Protein sequences
Rattus
Rodentia
Viral vectors
(Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Fusion proteins (chimeric proteins)
Nucleic acids
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Dimers
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo receptor-1 fusion protein; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Receptors
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo, 1; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Receptors
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Receptors

RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NogoA; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NogoB; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NogoC; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Glycoproteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(OMGP (oligodendrocyte myelin glycoprotein); Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Hybridoma
(PTA-4584-PTA-4588; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Nervous system, disease
(amyotrophic lateral sclerosis; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Biology
(cell, host; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chimeric; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Nerve, disease
(diabetic neuropathy; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fragments; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU

(Therapeutic use); BIOL (Biological study); USES (Uses)
(heavy chain; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(humanized; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(immunoadhesins; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Drug delivery systems
(immunoconjugates; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Nerve, disease
(inhibition; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Nerve, disease
(injury, inhibition; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Spinal cord, disease
(injury; Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT Proteins
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(leucine-rich repeat; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(light chain; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); PRP (Properties); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Cell death
(neuron, inhibition; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Central nervous system

(neuron; Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT Injury
(neuronal, inhibition; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Axon
Axon
(outgrowth, promotion; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Genetic element
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(signal sequence; Nogo receptor-1
antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT Injury
(spinal cord; Nogo receptor-1 antagonists
for promoting survival of neuron and treating multiple
sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT Brain, disease
(stroke; Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT Brain, disease
(trauma; Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT Ligands
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
('Nogo receptor-1; Nogo receptor
-1 antagonists for promoting survival of neuron and
treating multiple sclerosis, CNS neuropathy, and traumatic
brain or spinal cord injury)

IT 427799-82-4, GenBank AF462390
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL
(Biological study)
(Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT 662152-33-2 662152-34-3 662152-35-4 662152-36-5 662152-37-6
662152-38-7 662152-39-8 662152-40-1 662152-41-2 662152-42-3
662377-03-9 662377-04-0
RL: BSU (Biological study, unclassified); PRP (Properties); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo receptor-1 antagonists for
promoting survival of neuron and treating multiple sclerosis,
CNS neuropathy, and traumatic brain or spinal cord injury)

IT 662384-33-0P, 1-344-Nogo receptor 1 (human)
662384-34-1P, 1-310-Nogo receptor 1 (human)
662384-35-2P, 1-344-Nogo receptor 1 (rat)
662384-36-3P, 1-310-Nogo receptor 1 (rat)
662384-37-4P 662384-38-5P 662384-39-6P 663232-46-0P
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified);

PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; Nogo receptor-1 antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT 662385-15-1 662385-16-2 662385-17-3
RL: PRP (Properties)
(unclaimed nucleotide sequence; nogo receptor antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

IT 662397-79-7
RL: PRP (Properties)
(unclaimed sequence; nogo receptor antagonists for promoting survival of neuron and treating multiple sclerosis, CNS neuropathy, and traumatic brain or spinal cord injury)

L152 ANSWER 17 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:41639 HCAPLUS Full-text
DOCUMENT NUMBER: 140:106543
TITLE: Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases
INVENTOR(S): Orita, Satoshi; Shimazaki, Atsuyuki; Yanagimoto, Toru; Nakajima, Masatoshi; Oshima, Takeo
PATENT ASSIGNEE(S): Shionogi & Co., Ltd., Japan
SOURCE: PCT Int. Appl., 100 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2004005510	A1	20040115	WO 2003-JP8469		20030703 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2003246254	A1	20040123	AU 2003-246254		20030703 <--
PRIORITY APPLN. INFO.:			JP 2002-197188	A	20020705 <--
			WO 2003-JP8469	W	20030703 <--

ED Entered STN: 18 Jan 2004

AB This invention provides a novel protein 39C7 from human and rat, which has sequence homol. with Nogo receptor (NgR) family, encoding cDNA, recombinant expression, and drug screening, diagnostic, and therapeutic uses. A rat cDNA clone 39C7 coding for a Nogo receptor-like protein and a human homolog, were cloned. 39C7 showed elevated expression in the skeletal muscle of a diabetes model Zucker fatty rat having a restricted diet and taking exercises, upon improvement in insulin resistance. In a cell line overexpressing human 39C7,

glucose uptake was increased independent of insulin concentration The protein is useful as a diagnostic marker and a remedy for diabetes. Since this polypeptide is expressed most strongly in the cerebral cortex in the brain, it is also useful as a marker and a remedy for neurodegenerative diseases such as Alzheimer's disease.

IC ICM C12N015-09
ICS A01K067-027; A61K031-7088; A61K038-17; A61K039-395; A61K048-00; A61P003-10; A61P021-04; A61P025-00; A61P025-16; A61P025-28; C07K014-705; C07K016-28; C12P021-02; C12Q001-68; G01N033-15; G01N033-50; G01N033-53; G01N033-566

CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 6, 14

ST cDNA sequence Nogo receptor homolog 39C7 human rat; diabetes neurodegenerative disease diagnosis therapy

IT Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(39C7; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Repeat motifs (protein)
(LRR (leucine-rich repeat), presence of; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Antidiabetic agents
Biomarkers
Diabetes mellitus
Human
Molecular cloning
Nervous system agents
Protein sequences
Rattus
cDNA sequences
(Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Brain
(cerebral cortex, strong expression in; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Nervous system, disease
(degeneration; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Exercise
(diabetes model rat with, elevated expression in; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)

IT Disease models
(diabetes; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for

diabetes and neurodegenerative diseases)
IT Diagnosis
(genetic; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT Diagnosis
(immunodiagnosis; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT Diagnosis
(mol.; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT Muscle
(of diabetes model Zucker fatty rat, elevated expression in; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT Diet
(restricted, diabetes model rat with, elevated expression in; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT Biological transport
(uptake, glucose, insulin-independent increase in human 39C7 overexpressing cell line; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT 646074-87-5 646074-88-6
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT 646074-85-3 646074-86-4
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resistance, improvement in, diabetes model rat with, elevated expression in; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
IT 50-99-7, D-Glucose, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(uptake, insulin-independent increase in human 39C7 overexpressing cell line; Nogo receptor homolog protein 39C7 from human and rat and diagnostic, and therapeutic uses for diabetes and neurodegenerative diseases)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L152 ANSWER 18 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:513137 HCAPLUS Full-text
DOCUMENT NUMBER: 141:47360
TITLE: Inhibitors of myelin-associated glycoprotein (MAG) activity for regulating neural growth and regeneration

INVENTOR(S): Filbin, Marie T.; Domeniconi, Marco; Cao, Zixuan
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 81 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004121341	A1	20040624	US 2002-327213	20021220 <--
CA 2510297	A1	20040715	CA 2003-2510297	20031219 <--
WO 2004058169	A2	20040715	WO 2003-US40740	20031219 <--
WO 2004058169	A3	20050721		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003299756	A1	20040722	AU 2003-299756	20031219 <--
EP 1644016	A2	20060412	EP 2003-800036	20031219 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, SK

PRIORITY APPLN. INFO.:	US 2002-327213	A	20021220 <--
	WO 2003-US40740	W	20031219 <--

ED Entered STN: 25 Jun 2004
AB The present invention relates generally to products, compns. and methods useful for promoting neural repair and regeneration. The products and compns. of this invention include myelin-associated glycoprotein (MAG) derivs. that are inhibitors of endogenous MAG (e.g., mutant MAG proteins) and Nogo Receptor (NgR) binding inhibitors that are peptides derived from MAG, Nogo and OMgp that can bind to NgR and block NgR signaling. Peptides that can bind and activate NgR signaling are also provided. Inhibitory MAG derivs. and NgR binding inhibitors are useful for blocking the inhibition of neural regeneration mediated by proteins such as MAG, Nogo and/or OMgp in the nervous system. These inhibitors are also useful for treating neural degeneration associated with injuries, disorders or diseases.
IC ICM C12Q001-68
ICS C07H021-04; C07K014-47
INCL 435006000; 435069100; 435320100; 435325000; 530395000; 536023500
CC 1-11 (Pharmacology)
IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo, Nogo receptor-binding inhibitors; inhibitors of myelin-associated glycoprotein (MAG) activity for regulating neural growth and regeneration)
IT Alzheimer's disease
Aneurysm
Anti-Alzheimer's agents
Antiparkinsonian agents
Drug delivery systems
Multiple sclerosis
Nerve regeneration
Nerve regeneration

Nervous system agents
Parkinson's disease
Prion diseases
Protein sequences
(inhibitors of myelin-associated glycoprotein (MAG) activity for regulating neural growth and regeneration)

L152 ANSWER 19 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:1006693 HCAPLUS Full-text
DOCUMENT NUMBER: 140:58448
TITLE: Antigen-presenting cells for neuroprotection and nerve regeneration
INVENTOR(S): Eisenbach-Schwartz, Michal; Cohen, Avraham
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel
SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003105750	A2	20031224	WO 2003-IL500	20030612 <--
WO 2003105750	A3	20050728		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2488855	A1	20031224	CA 2003-2488855	20030612 <--
AU 2003231909	A1	20031231	AU 2003-231909	20030612 <--
EP 1578199	A2	20050928	EP 2003-760117	20030612 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1705438	A	20051207	CN 2003-819321	20030612 <--
JP 2006503808	T	20060202	JP 2004-512658	20030612 <--
US 2006057110	A1	20060316	US 2004-517666	20041213 <--
PRIORITY APPLN. INFO.:			US 2002-388296P P	20020614 <--
			WO 2003-IL500 W	20030612 <--

ED Entered STN: 26 Dec 2003
AB The authors disclose pharmaceutical compns. and methods for preventing or inhibiting neuronal degeneration, or for promoting nerve regeneration, in the central nervous system (CNS) or peripheral nervous system (PNS), in the treatment of an injury, disorder or disease of the CNS or PNS. The treatment comprises antigen-presenting cells, preferably dendritic cells, that have been pulsed with an agent selected from the group consisting of: (a) a nervous system (NS)-specific antigen or an analog thereof; (b) a peptide or altered peptide ligand derived from an NS-specific antigen; (c) a copolymer selected from the group consisting of Copolymer 1, a Copolymer 1-related peptide or polypeptide, and poly-Glu50 Tyr50; and (d) a non-self antigen. In one example, local implantation of bone marrow-derived dendritic cells exposed to MBP peptide promoted functional recovery in a spinal cord contusion model.
IC ICM A61K
CC 15-8 (Immunochemistry)

Section cross-reference(s): 1, 2, 14
IT Receptors
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; with antigen-presenting cells for elicitation of T-cell-dependent neuroprotection and nerve regeneration in nervous system injury)
IT Alzheimer's disease
Amnesia
Anxiety
Central nervous system, disease
Epilepsy
Glaucoma (disease)
Oxidative stress, biological
Parkinson's disease
Peripheral nervous system, disease
(antigen-presenting cells for elicitation of antigen- and T-cell-dependent neuroprotection and nerve regeneration in)
IT Amyloid
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(β-; with antigen-presenting cells for elicitation of T-cell-dependent neuroprotection and nerve regeneration in nervous system injury)

L152 ANSWER 20 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:855771 HCAPLUS Full-text
DOCUMENT NUMBER: 139:345937
TITLE: BACE1 regulation by RTN3 and RTN4 and methods for drug screening and treating amyloidoses
INVENTOR(S): Yan, Riqiang; Lu, Yifeng
PATENT ASSIGNEE(S): Pharmacia & Upjohn Company, USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003088926	A2	20031030	WO 2003-US8829	20030408 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2482589	A1	20031030	CA 2003-2482589	20030408 <--
AU 2003223330	A1	20031103	AU 2003-223330	20030408 <--
US 2004063161	A1	20040401	US 2003-408967	20030408 <--
EP 1575482	A2	20050921	EP 2003-719447	20030408 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006500006	T	20060105	JP 2003-585679	20030408 <--
BR 2003009110	A	20070508	BR 2003-9110	20030408 <--

MX 2004PA09498 A 20050517 MX 2004-PA9498 20040929 <--
PRIORITY APPLN. INFO.: US 2002-373284P P 20020417 <--
WO 2003-US8829 W 20030408 <--

ED Entered STN: 31 Oct 2003

AB The invention relates to compns. and methods for treating Alzheimer's Disease and other amyloidoses, to polypeptides that modulate BACE1 activity, and methods to identify agents for use in treating Alzheimer's Disease and other amyloidoses. This invention is based, in part, on the novel finding that RTN3 or RTN4 modulates the activity of BACE1. Thus, in one aspect, the invention provides a method of modulating BACE1 activity in a humans and animals by administration of an exogenous RTN3 or exogenous RTN4 polypeptide or administration of one or more agents that affect the expression or activity of endogenous RTN3 or RTN4. Polypeptides that are derived from RTN3 sequence and possess one or more function or biol. activities of RTN3, polynucleotide sequences encoding the recombinant polypeptides, and method of making the recombinant polypeptides are also included. Also included are in vitro or in vivo methods to identify agents that modulate (1) the expression or activity of RTN3 or RTN4 or (2) the activity of BACE1, agents modulating the activity of BACE1 where the said agents are exogenous RTN3, exogenous RTN4 polypeptide, recombinant polypeptides of the invention, and agents that affect the expression or activity of endogenous RTN3 or RTN4.

IC ICM A61K

CC 1-11 (Pharmacology)
Section cross-reference(s): 3

ST amyloidosis Alzheimer disease treatment RTN3 RTN4 BACE1

IT Alzheimer's disease
Anti-Alzheimer's agents
Drug screening
Human
(Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Proteins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(Nogo; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Gene, animal
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(RTN3, modulators of expression of; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Animal cell line
Animal tissue culture
(RTN3/RTN4-expressing, drug screening with; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Brain, disease
(amyloid angiopathy; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Myositis
(inclusion body, sporadic; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Amyloidosis
(inhibitors of; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Molecular association
(of BACE1 with RTN3/RTN4 proteins; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Molecular cloning

(of RTN3/RTN4 genes; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Proteins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(reticulon RTN3; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT Amyloid
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(β-, modulators of production of; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT 158736-49-3, Proteinase BACE1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT 618464-17-8P, Reticulon (human gene RTN3) 618472-87-0P, Nogo protein (human gene RTN4 isoform B) 618472-88-1P, Nogo protein (human gene RTN4 isoform C)
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT 618464-18-9P
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT 618464-16-7, DNA (human gene RTN3 reticulon cDNA)
RL: BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); USES (Uses)
(nucleotide sequence; Alzheimer's disease treatment with polypeptides modulating BACE1 activity)

IT 618519-07-6 618519-08-7 618519-10-1
RL: PRP (Properties)
(unclaimed nucleotide sequence; bACE1 regulation by RTN3 and RTN4 and methods for drug screening and treating amyloidoses)

IT 618519-09-8
RL: PRP (Properties)
(unclaimed protein sequence; bACE1 regulation by RTN3 and RTN4 and methods for drug screening and treating amyloidoses)

L152 ANSWER 21 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:946440 HCAPLUS Full-text
DOCUMENT NUMBER: 138:38058
TITLE: Human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers
INVENTOR(S): Anderson, David W.; Zerhusen, Bryan D.; Li, Li; Zhong, Mei; Casman, Stacie J.; Gerlach, Valerie L.; Shimkets, Richard A.; Gorman, Linda; Pena, Carol E. A.; Kekuda, Ramesh; Patturajan, Meera; Spytek, Kimberly A.; Leite, Mario W.; Rastelli, Luca; MacDougall, John R.; Taupier, Raymond J., Jr.; Guo, Xiaojia; Miller, Charles E.; Shenoy, Suresh G.; Hjalt, Tord; Voss, Edward Z.; Boldog, Ferenc L.; Malyankar, Uriel M.;

Padigaru, Muralidhara; Ji, Weizhen; Smithson, Glennda; Edinger, Shlomit R.; Millet, Isabelle; Ellerman, Karen

PATENT ASSIGNEE(S): Curagen Corporation, USA

SOURCE: PCT Int. Appl., 461 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 175

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099062	A2	20021212	WO 2002-US17559	20020604 <--
WO 2002099062	A3	20030220		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR; LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018555	A1	20040129	US 2002-161493	20020603 <--
CA 2447935	A1	20021212	CA 2002-2447935	20020604 <--
AU 2002303960	A1	20021216	AU 2002-303960	20020604 <--
EP 1401470	A2	20040331	EP 2002-732027	20020604 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2005506833	T	20050310	JP 2003-502172	20020604 <--
EP 1661998	A2	20060531	EP 2006-2417	20020604 <--
EP 1661998	A3	20061206		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
AU 2005200106	A1	20050210	AU 2005-200106	20050112 <--
US 2006063200	A1	20060323	US 2005-51724	20050202 <--
US 2005266431	A1	20051201	US 2005-64246	20050222 <--
AU 2006201467	A1	20060504	AU 2006-201467	20060407 <--
AU 2007202935	A1	20070719	AU 2007-202935	20070626 <--

PRIORITY APPLN. INFO.:

US 2001-295607P	P	20010604 <--
US 2001-296404P	P	20010606 <--
US 2001-296418P	P	20010606 <--
US 2001-296575P	P	20010607 <--
US 2001-297414P	P	20010611 <--
US 2001-297567P	P	20010612 <--
US 2001-297573P	P	20010612 <--
US 2001-298285P	P	20010614 <--
US 2001-298528P	P	20010615 <--
US 2001-298556P	P	20010615 <--
US 2001-299133P	P	20010618 <--
US 2001-299230P	P	20010619 <--
US 2001-299949P	P	20010621 <--
US 2001-300177P	P	20010622 <--
US 2001-301530P	P	20010628 <--
US 2001-301550P	P	20010628 <--
US 2001-302951P	P	20010703 <--
US 2001-318771P	P	20010912 <--
US 2001-324687P	P	20010925 <--
US 2001-339266P	P	20011024 <--
US 2001-337524P	P	20011116 <--

US 2001-341143P	P	20011214 <--
US 2002-358643P	P	20020221 <--
US 2002-359151P	P	20020221 <--
US 2002-361195P	P	20020228 <--
US 2002-361964P	P	20020305 <--
US 2002-371346P	P	20020410 <--
US 2002-371523P	P	20020410 <--
US 2002-161493	A2	20020603 <--
AU 2000-37360	A3	20000309 <--
AU 2000-78680	A3	20001006 <--
AU 2001-247294	A3	20010305 <--
AU 2001-47294	T0	20010305 <--
US 2001-295661P	P	20010604 <--
US 2001-300290P	P	20010622 <--
US 2001-300883P	P	20010626 <--
US 2001-311285P	P	20010809 <--
US 2001-311972P	P	20010813 <--
US 2001-315069P	P	20010827 <--
US 2001-315071P	P	20010827 <--
US 2001-315660P	P	20010829 <--
US 2001-322293P	P	20010914 <--
US 2001-322706P	P	20010917 <--
US 2001-325687P	P	20010928 <--
US 2001-327345P	P	20011005 <--
US 2001-327892P	P	20011009 <--
US 2001-341186P	P	20011214 <--
US 2002-361189P	P	20020228 <--
US 2002-363673P	P	20020312 <--
US 2002-363676P	P	20020312 <--
US 2002-161493P	P	20020603 <--
US 2002-162335	A1	20020603 <--
EP 2002-732027	A3	20020604 <--
WO 2002-US17559	W	20020604 <--
US 2002-177809	B1	20020621 <--

ED Entered STN: 13 Dec 2002

AB Disclosed herein are nucleic acid sequences that encode NOVX polypeptides such as NOV1, NOV2, NOV3, etc.. Also disclosed are antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, prognosis, treatment, and prevention of human diseases involving any one of these novel human nucleic acids, polypeptides, or antibodies, or fragments thereof, such as cancer.

IC ICM C12N

CC 15-2 (Immunochemistry)
Section cross-reference(s): 1, 3, 9, 63

ST human NOVX protein polynucleotide antibody cancer diagnosis prognosis therapy

IT Claudins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (9; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (C4bp (complement C4b-binding protein); human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT CD antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(CD53; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Inflammation
(Crohn's disease; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Intestine, disease
(Crohn's; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ebnerin; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Tumor antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MEA6/11; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV10; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV11; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV12; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV13; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation);

BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV14; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV15; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV16; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV17; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV18; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV19; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV1; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV20; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV21; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV22; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV23; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV24; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV25; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)

(NOV26; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV27; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV28; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV29; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV2; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV30; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV31; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation);

BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV32; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV33; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV34; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV35; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV36; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV37; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV38; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV39; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV3; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV40; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV41; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV42; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV43; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)

(NOV44; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV45; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV46; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV4; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV5; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV6; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV7; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation);

BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV8; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOV9; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Gene, animal
Proteins
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(NOVX; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Tm7; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Tumor antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(XAGE; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Diagnosis
Diagnosis
(cancer; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Drug delivery systems
(carriers; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cellular; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Test kits
(diagnostic; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Biomarkers
(disease; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders

and cancers)

IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(endosomal precursor; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fragments; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(fusion products; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(histidine transporter; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Alzheimer's disease
Animals
Antitumor agents
Asthma
DNA sequences
Diabetes mellitus
Diagnosis
Dissociation constant
Drug screening
Eubacteria
Genetic vectors
Human
Immunotherapy
Inflammation
Insecta
Mammalia
Metabolic disorders
Molecular cloning
Nucleic acid hybridization
Prognosis
Protein sequences
Susceptibility (genetic)
Yeast
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Transgene
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis,

prognosis and therapy of NOVX-associated disorders and cancers)

IT Cadherins
Cadherins
Prion proteins
Promoter (genetic element)
Syntaxins
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Primers (nucleic acid)
Probes (nucleic acid)
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); BIOL (Biological study); USES (Uses)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Natural products, pharmaceutical
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Disease, animal
(human NOVX-associated; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(humanized; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Diagnosis
(immunodiagnosis; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Animal cell
(mammalian; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Tumor antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(mammary Mat8; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Animal cell
Animal tissue
(marker; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(membrane, integral; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(membrane, type III; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Nervous system, neoplasm
(meningioma, antigen; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Neoplasm
(metastasis; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Meninges
(neoplasm, meningioma, antigen; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(neurofascin precursor; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Nerve, disease
(neuropathy; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Antibodies and Immunoglobulins
RL: ARG (Analytical reagent use); BPN (Biosynthetic preparation); DGN (Diagnostic use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(neutralizing; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(phospholipid transporter; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proline transporter; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Cadherins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(protocadherin, α C1-like; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(sialic acid-binding; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Agglutinins and Lectins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(siglec; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT Mutagenesis
(site-directed, substitution; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(surface, leukocyte; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Proteins
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(test; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(transmembrane; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Injury
(trauma; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Thrombospondins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(type 1 motif; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Inflammation
Intestine, disease
(ulcerative colitis; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Integrins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(α8; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT Transforming growth factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β-, binding protein 3; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)
IT 478431-29-7P, Protein NOV1a (human) 478431-31-1P, Protein NOV2a (human) 478431-33-3P, Protein NOV3a (human) 478431-35-5P, Protein NOV4a (human) 478431-37-7P, Protein NOV5a (human) 478431-39-9P, Protein NOV6a (human) 478431-41-3P, Protein NOV6b (human) 478431-43-5P, Protein NOV7a (human) 478431-45-7P, Protein NOV8a (human) 478431-47-9P, Protein NOV9a (human) 478431-49-1P, Protein NOV9b (human) 478431-51-5P, Protein NOV10a (human) 478431-53-7P, Protein NOV11a (human) 478431-55-9P, Protein NOV12a (human) 478431-57-1P, Protein NOV12b (human) 478431-59-3P, Protein NOV13a (human) 478431-61-7P, Protein NOV13b (human) 478431-63-9P, Protein NOV14a (human) 478431-65-1P, Protein NOV15a (human) 478431-67-3P, Protein NOV15b (human) 478431-70-8P, Protein NOV17a (human) 478431-72-0P, Protein NOV17b (human) 478431-74-2P, Protein NOV17c (human) 478431-76-4P, Protein NOV18a (human) 478431-78-6P, Protein NOV19a (human) 478431-80-0P, Protein NOV20a (human) 478431-82-2P, Protein NOV20b (human) 478431-84-4P, Protein NOV21a (human) 478431-86-6P, Protein NOV22a (human) 478431-88-8P, Protein

NOV23a (human) 478431-90-2P, Protein NOV24a (human) 478431-92-4P, Protein NOV24b (human) 478431-94-6P, Protein NOV25a (human) 478431-96-8P, Protein NOV26a (human) 478431-98-0P, Protein NOV26b (human) 478432-00-7P, Protein NOV27a (human) 478432-02-9P, Protein NOV28a (human) 478432-04-1P, Protein NOV29a (human) 478432-06-3P, Protein NOV30a (human) 478432-08-5P, Protein NOV30b (human) 478432-10-9P, Protein NOV30c (human) 478432-12-1P, Protein NOV30d (human) 478432-14-3P, Protein NOV30e (human) 478432-16-5P, Protein NOV31a (human) 478432-18-7P, Protein NOV31b (human) 478432-20-1P, Protein NOV31c (human) 478432-22-3P, Protein NOV32a (human) 478432-24-5P, Protein NOV33a (human) 478432-27-8P, Protein NOV36a (human) 478432-29-0P, Protein NOV37a (human) 478432-31-4P, Protein NOV37b (human) 478432-33-6P, Protein NOV37c (human) 478432-35-8P, Protein NOV38a (human) 478432-37-0P, Protein NOV38b (human) 478432-39-2P, Protein NOV39a (human) 478432-41-6P, Protein NOV39b (human) 478432-43-8P, Protein NOV39c (human) 478432-45-0P, Protein NOV39d (human) 478432-47-2P, Protein NOV39e (human) 478432-49-4P, Protein NOV39f (human) 478432-51-8P, Protein NOV40a (human) 478432-53-0P, Protein NOV40b (human) 478432-55-2P, Protein NOV40c (human) 478432-57-4P, Protein NOV40d (human) 478432-59-6P, Protein NOV40e (human) 478432-61-0P, Protein NOV41a (human) 478432-62-1P, Protein NOV34a (human) 478432-64-3P, Protein NOV35a (human) 478432-66-5P, Protein NOV35b (human) 478432-69-8P, Protein NOV35c (human) 478432-71-2P, Protein NOV35d (human) 478432-72-3P, Protein NOV35e (human) 478432-73-4P, Protein NOV16a (human)
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT 9000-83-3, ATPase 9028-86-8, Aldehyde dehydrogenase 37259-58-8, Serine protease 40581-18-8, Emerin 109319-16-6 153190-52-4, Colon carcinoma kinase 4 171715-27-8, Reprolysin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT 478431-28-6P 478431-30-0P 478431-32-2P 478431-34-4P 478431-36-6P
478431-38-8P 478431-40-2P 478431-42-4P 478431-44-6P 478431-46-8P
478431-48-0P 478431-50-4P 478431-52-6P 478431-54-8P 478431-56-0P
478431-58-2P 478431-60-6P 478431-62-8P 478431-64-0P 478431-66-2P
478431-68-4P 478431-69-5P 478431-71-9P 478431-73-1P, DNA (human protein NOV17c cDNA) 478431-75-3P 478431-77-5P 478431-79-7P
478431-81-1P 478431-83-3P 478431-85-5P 478431-87-7P 478431-89-9P
478431-91-3P 478431-93-5P 478431-95-7P, ADNA (human protein NOV26a cDNA) 478431-97-9P, DNA (human protein NOV26b cDNA) 478431-99-1P
478432-01-8P 478432-03-0P 478432-05-2P 478432-07-4P 478432-09-6P, DNA (human protein NOV30c cDNA) 478432-11-0P, DNA (human protein NOV30d cDNA) 478432-13-2P, DNA (human protein NOV30e cDNA) 478432-15-4P, DNA (human protein NOV31a cDNA) 478432-17-6P, DNA (human protein NOV31b cDNA) 478432-19-8P 478432-21-2P 478432-23-4P 478432-25-6P
478432-26-7P 478432-28-9P 478432-30-3P 478432-32-5P 478432-34-7P, DNA (human protein NOV38a cDNA) 478432-36-9P, DNA (human protein NOV38b cDNA) 478432-38-1P 478432-40-5P, DNA (human protein NOV39b cDNA)
478432-42-7P, DNA (human protein NOV39c cDNA) 478432-44-9P, DNA (human protein NOV39d cDNA) 478432-46-1P, DNA (human protein NOV39e cDNA)
478432-48-3P, DNA (human protein NOV39f cDNA) 478432-50-7P
478432-52-9P, DNA (human protein NOV40b cDNA) 478432-54-1P
478432-56-3P, DNA (human protein NOV40d cDNA) 478432-58-5P, DNA (human

protein NOV40e cDNA) 478432-60-9P, DNA (human protein NOV41a cDNA) 478432-63-2P 478432-65-4P, DNA (human protein NOV35b cDNA) 478432-67-6P, DNA (human protein NOV35c cDNA) 478432-68-7P, DNA (human protein NOV35d cDNA) 478432-70-1P, DNA (human protein NOV35e cDNA)
RL: ARU (Analytical role, unclassified); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT 478452-21-0 478452-22-1 478452-23-2 478452-24-3 478452-25-4
478452-26-5 478452-27-6 478452-28-7 478452-29-8 478452-30-1
478452-31-2 478452-32-3 478452-33-4 478452-34-5 478452-35-6
478452-36-7 478452-37-8 478452-38-9 478452-39-0 478452-40-3
478452-41-4 478452-42-5 478452-43-6 478452-44-7 478452-45-8
478452-46-9 478452-47-0 478452-48-1 478452-49-2 478452-50-5
478452-51-6 478452-52-7 478452-53-8 478452-54-9 478452-55-0
478452-56-1 478452-57-2 478452-58-3 478452-59-4 478452-60-7
478452-61-8 478452-62-9 478452-63-0 478452-64-1 478452-65-2
478452-66-3 478452-67-4 478452-68-5 478452-69-6 478452-70-9
478452-71-0 478452-72-1 478452-73-2 478452-74-3 478452-75-4
478452-76-5 478452-77-6 478452-78-7 478452-79-8 478452-80-1
478452-81-2 478452-82-3 478452-83-4 478452-84-5 478452-85-6
478452-86-7 478452-87-8 478452-88-9 478452-89-0 478452-90-3
478452-91-4 478452-92-5 478452-93-6 478452-94-7 478452-95-8
478452-96-9 478452-97-0 478452-98-1 478452-99-2 478453-00-8
478453-01-9 478453-02-0 478453-03-1 478453-04-2 478453-05-3
478453-06-4 478453-07-5 478453-08-6 478453-09-7 478453-10-0
478453-11-1 478453-12-2 478453-13-3 478453-14-4 478453-15-5
478453-16-6 478453-17-7 478453-18-8 478453-19-9 478453-20-2
478453-21-3 478453-22-4 478453-23-5 478453-24-6 478453-25-7
478453-26-8 478453-27-9 478453-28-0 478453-29-1 478453-30-4
478453-31-5 478453-32-6 478453-33-7 478453-34-8 478453-35-9
478453-36-0 478453-37-1 478453-38-2 478453-39-3 478453-42-8
478453-47-3 478453-52-0 478453-53-1 478453-54-2 478453-55-3
478453-56-4 478453-57-5 478453-58-6 478453-59-7 478453-60-0
478453-61-1 478453-62-2 478453-63-3 478453-64-4 478453-65-5
RL: PRP (Properties)
(unclaimed nucleotide sequence; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

IT 478453-40-6 478453-41-7 478453-43-9 478453-44-0 478453-45-1
478453-46-2 478453-48-4 478453-49-5 478453-50-8 478453-51-9
RL: PRP (Properties)
(unclaimed sequence; human NOVX polypeptides, polynucleotides and antibodies for diagnosis, prognosis and therapy of NOVX-associated disorders and cancers)

L152 ANSWER 22 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:450338 HCAPLUS Full-text
DOCUMENT NUMBER: 137:32058
TITLE: Nervous system-specific antigens and activated T cells for neuroprotection and neuronal degeneration inhibition
INVENTOR(S): Eisenbach-Schwartz, Michal; Hauben, Ehud; Cohen, Irun R.; Beserman, Pierre; Mosonego, Alon; Moalem, Gila
PATENT ASSIGNEE(S): Yeda Research and Development Co. Ltd., Israel
SOURCE: U.S. Pat. Appl. Publ., 93 pp., Cont.-in-part of U.S. Ser. No. 314,161.

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002072493	A1	20020613	US 2001-893348	20010628 <--
WO 9934827	A1	19990715	WO 1998-US14715	19980721 <--
W: AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GE, HR, HU, ID, IL, IS, JP, KG, KP, KR, KZ, LC, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TJ, TM, TR, TT, UA, US, UZ, VN, YU				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2003108528	A1	20030612	US 1998-218277	19981222 <--
WO 2003002602	A2	20030109	WO 2002-IL518	20020627 <--
WO 2003002602	A3	20031023		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002314509	A1	20030303	AU 2002-314509	20020627 <--
US 2004253218	A1	20041216	US 2004-810653	20040329 <--

PRIORITY APPLN. INFO.:			
IL 1998-124500	A	19980519	<--
WO 1998-US14715	A2	19980721	<--
US 1998-218277	A2	19981222	<--
US 1999-314161	A2	19990519	<--
IL 1998-124550	A	19980519	<--
US 2001-893348	A	20010628	<--
WO 2002-IL518	W	20020627	<--

ED Entered STN: 14 Jun 2002

AB Compns. and methods to promote nerve regeneration or to confer neuroprotection and prevent or inhibit neuronal degeneration within the nervous system, either the central nervous system or the peripheral nervous system, are provided. Treatment involves administering NS-specific activated T cells, or an NS-specific antigen or analog thereof, a peptide derived therefrom or an analog or derivative of said peptide, or a nucleotide sequence encoding said antigen or peptide, or any combination thereof. The NS-specific antigen is myelin basic protein, myelin oligodendrocyte glycoprotein, proteolipid, myelin-associated glycoprotein, S-100, β-amyloid, Thy-1, P0, P2 or neurotransmitter receptor.

IC ICM A61K038-17
INCL 514012000
CC 15-2 (Immunochemistry)
Section cross-reference(s): 1, 3, 63

IT Antigens
Receptors
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Nogo; nervous system-specific antigens and activated T cells for neuroprotection and neuronal degeneration inhibition)

IT Alzheimer's disease
Central nervous system, disease
DNA sequences
Fabry disease
Glaucoma (disease)
Human
Mus
Nervous system, disease
Parkinson's disease
Peripheral nervous system, disease
Prion diseases
Protein sequences
Rattus
Refsum disease
Surgery
T cell (lymphocyte)
(nervous system-specific antigens and activated T cells for neuroprotection and neuronal degeneration inhibition)

IT Amyloid
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(β-; nervous system-specific antigens and activated T cells for neuroprotection and neuronal degeneration inhibition)

IT 437135-40-5, Myelin basic protein (human gene MBP) 437135-48-3, Proteolipid protein (human gene PLP) 437135-49-4 437135-54-1, Protein NogoA (Rattus norvegicus) 437135-55-2, Protein NogoB (Rattus norvegicus) 437135-56-3, Protein NogoC (Rattus norvegicus) 437135-57-4, Protein NogoA (human) 437135-58-5, Protein NogoB (human) 437135-59-6, Protein NogoC (human) 437135-60-9, Protein Nogo receptor (human) 437135-61-0, Protein Nogo receptor (Mus musculus)
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; nervous system-specific antigens and activated T cells for neuroprotection and neuronal degeneration inhibition)

L152 ANSWER 23 OF 36 HCAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:694987 HCAPLUS Full-text
DOCUMENT NUMBER: 137:350109
TITLE: Modulation of axonal regeneration in neurodegenerative disease. Focus on Nogo
AUTHOR(S): Strittmatter, Stephen M.
CORPORATE SOURCE: Department of Neurology, and Section of Neurobiology, Yale University School of Medicine, New Haven, CT, 06510, USA
SOURCE: Journal of Molecular Neuroscience (2002), 19(1/2), 117-121
CODEN: JMNEES; ISSN: 0895-8696
PUBLISHER: Humana Press Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ED Entered STN: 13 Sep 2002

AB A review. Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears to have a physiol. role in regulating structural plasticity. The possibility that the Nogo system contributes to pathol. and compensatory plasticity in Alzheimer's Disease is considered.

CC 14-0 (Mammalian Pathological Biochemistry)
ST review Nogo receptor axon regeneration neurodegeneration Alzheimer

IT Alzheimer's disease
Axon
Nerve regeneration
Nerve regeneration
Synaptic plasticity
(Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)
IT Proteins
Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Nogo; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)
IT Nervous system, disease
(degeneration; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)
REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d L152 24-36 ibib ab hit

L152 ANSWER 24 OF 36 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2003258477 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12784033
TITLE: Unaltered plasma levels of beta-amyloid(1-40) and beta-amyloid(1-42) upon stimulation of human platelets.
AUTHOR: Olsson Annika; Vanmechelen Eugeen; Vanderstichele Hugo; Davidsson Pia; Blennow Kaj
CORPORATE SOURCE: Institute of Clinical Neuroscience, Experimental Neuroscience Section, Goteborg University, Sahlgrenska University Hospital/Molndal, Molndal, Sweden.. Annika.Olsson@neuro.gu.se
SOURCE: Dementia and geriatric cognitive disorders, (2003) Vol. 16, No. 2, pp. 93-7.
Journal code: 9705200. ISSN: 1420-8008.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200310
ENTRY DATE: Entered STN: 5 Jun 2003
Last Updated on STN: 16 Oct 2003
Entered Medline: 15 Oct 2003

AB Accumulation of beta-amyloid (Abeta) in the brain is one of the central lesions in Alzheimer's disease (AD). Alternative cleavage of the amyloid precursor protein (APP), occurring in both normal and AD subjects, results in the generation and secretion of soluble APP, Abeta(40) and Abeta(42). Platelets have been regarded as the primary source of circulating APP and Abeta. Plasma levels of Abeta may therefore be dependent on platelet activation. We analysed Abeta(40/42) in plasma in the presence of physiological agonists of platelet activation such as adenosine diphosphate, collagen, thrombin, and a synthetic agonist , thrombin receptor activator peptide 6. We found that the levels of Abeta(40/42) in plasma were not related to platelet activation, suggesting that sampling techniques affecting platelet activation do not confound measurement of Abeta(40/42)in plasma. Copyright 2003 S. Karger AG, Basel
SO Dementia and geriatric cognitive disorders, (2003) Vol. 16, No. 2, pp. 93-7.
Journal code: 9705200. ISSN: 1420-8008.

AB Accumulation of beta-amyloid (Abeta) in the brain is one of the central lesions in Alzheimer's disease (AD). Alternative cleavage of the amyloid precursor protein (APP), occurring in both normal and AD subjects, results in the generation and secretion of soluble APP, Abeta(40) and Abeta(42). Platelets have been regarded as the primary source of circulating APP and Abeta. Plasma levels of Abeta may therefore be dependent on platelet activation. We analysed Abeta(40/42) in plasma in the presence of physiological agonists of platelet activation such as adenosine diphosphate, collagen, thrombin, and a synthetic agonist , thrombin receptor activator peptide 6. We found that the levels of Abeta(40/42) in plasma were not related to platelet activation, suggesting that sampling techniques affecting platelet activation do not confound measurement of Abeta(40/42)in plasma. Copyright 2003 S. Karger AG, Basel
CT Alzheimer Disease
Amyloid Precursor Protein Secretases
*Amyloid beta-Protein: BL, blood
Aspartic Endopeptidases: BL, blood
*Blood Platelets: ME, metabolism
Endopeptidases
Humans
Membrane Proteins: BL, blood
*Peptide Fragments: BL, blood
Platelet Activation
Presenilin-1
CN 0 (Amyloid beta-Protein); 0 (Membrane Proteins); 0 (PSEN1 protein, human); 0 (Peptide Fragments); 0 (Presenilin-1); 0 (amyloid beta-protein (1-40)); 0 (amyloid beta-protein (1-42)); EC 3.4.- (Amyloid Precursor Protein Secretases); EC 3.4.- (Endopeptidases); EC 3.4.23.- (Aspartic Endopeptidases); EC 3.4.23.46 (BACE1 protein, human)

L152 ANSWER 25 OF 36 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2002346485 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12088742
TITLE: Uptake and pathogenic effects of amyloid beta peptide 1-42 are enhanced by integrin antagonists and blocked by NMDA receptor antagonists.
AUTHOR: Bi X; Gall C M; Zhou J; Lynch G
CORPORATE SOURCE: Psychiatry and Human Behavior, 101 Theory, Suite 250, University of California at Irvine, 92697, USA.. xbi@uci.edu
CONTRACT NUMBER: AG00538 (NIA)
NS37799 (NINDS)
SOURCE: Neuroscience, (2002) Vol. 112, No. 4, pp. 827-40.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 29 Jun 2002
Last Updated on STN: 4 Sep 2002
Entered Medline: 3 Sep 2002

AB Many synapses contain two types of receptors - integrins and N-methyl-D-aspartate (NMDA) receptors - that have been implicated in peptide internalization. The present studies tested if either class is involved in

the uptake of the 42-residue form of amyloid beta peptide (Abeta1-42), an event hypothesized to be of importance in the development of Alzheimer's disease. Cultured hippocampal slices were exposed to Abeta1-42 for 6 days in the presence or absence of soluble Gly-Arg-Gly-Asp-Ser-Pro, a peptide antagonist of Arg-Gly-Asp (RGD)-binding integrins, or the disintegrin echistatin. Abeta uptake, as assessed with immunocytochemistry, occurred in 42% of the slices incubated with Abeta peptide alone but in more than 80% of the slices co-treated with integrin antagonists.' Uptake was also found in a broader range of hippocampal subfields in RGD-treated slices. Increased sequestration was accompanied by two characteristics of early stage Alzheimer's disease: elevated concentrations of cathepsin D immunoreactivity and activation of microglia. The selective NMDA receptor antagonist D-(-)-2-amino-5-phosphonovalerate completely blocked internalization of Abeta, up-regulation of cathepsin D, and activation of microglia. Our results identify two classes of receptors that cooperatively regulate the internalization of Abeta1-42 and support the hypothesis that characteristic pathologies of Alzheimer's disease occur once critical intraneuronal Abeta concentrations are reached.

TI Uptake and pathogenic effects of amyloid beta peptide 1-42 are enhanced by integrin antagonists and blocked by NMDA receptor antagonists.

SO Neuroscience, (2002) Vol. 112, No. 4, pp. 827-40.
Journal code: 7605074. ISSN: 0306-4522.

AB Many synapses contain two types of receptors - integrins and N-methyl-D-aspartate (NMDA) receptors - that have been implicated in peptide internalization. The present studies tested if either class is involved in the uptake of the 42-residue form of amyloid beta peptide (Abeta1-42), an event hypothesized to be of importance in the development of Alzheimer's disease. Cultured hippocampal slices were exposed to Abeta1-42 for 6 days in the presence or absence of soluble Gly-Arg-Gly-Asp-Ser-Pro, a peptide antagonist of Arg-Gly-Asp (RGD)-binding integrins, or the disintegrin echistatin. Abeta uptake, as assessed with immunocytochemistry, occurred in 42% of the slices incubated with Abeta peptide alone but in more than 80% of the slices co-treated with integrin antagonists. Uptake was also found in a broader range of hippocampal subfields in RGD-treated slices. Increased sequestration was accompanied by two characteristics of early stage Alzheimer's disease: elevated concentrations of cathepsin D immunoreactivity and activation of microglia. The selective NMDA receptor antagonist D-(-)-2-amino-5-phosphonovalerate completely blocked internalization of Abeta, up-regulation of cathepsin D, and activation of microglia. Our results identify two classes of receptors that cooperatively regulate the internalization of Abeta1-42 and support the hypothesis that characteristic pathologies of Alzheimer's disease occur once critical intraneuronal Abeta concentrations are reached.

CT 2-Amino-5-phosphonovalerate: PD, pharmacology
Alzheimer Disease: ME, metabolism
*Amyloid beta-Protein: AE, adverse effects
*Amyloid beta-Protein: ME, metabolism
Animals
Cathepsin D: ME, metabolism
Culture Techniques
*Hippocampus: ME, metabolism
Immunohistochemistry
*Integrins: AI, antagonists & inhibitors
*Integrins: ME, metabolism
Microglia: ME, metabolism
*Oligopeptides: PD, pharmacology
*Peptide Fragments: AE, adverse effects
*Peptide Fragments: ME, metabolism
Rats

Rats, Sprague-Dawley
*Receptors, N-Methyl-D-Aspartate: AI, antagonists & inhibitors
*Receptors, N-Methyl-D-Aspartate: ME, metabolism

CN 0 (Amyloid beta-Protein); 0 (Integrins); 0 (Oligopeptides); 0 (Peptide Fragments); 0 (Receptors, N-Methyl-D-Aspartate); 0 (amyloid beta-protein (1-42)); 0 (glycyl-arginyl-alanyl-aspartyl-seryl-proline); EC 3.4.23.5 (Cathepsin D)

L152 ANSWER 26 OF 36 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 94357904 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 8077213
TITLE: Thrombin receptor activation induces secretion and nonamyloidogenic processing of amyloid beta-protein precursor.
AUTHOR: Davis-Salinas J; Saporito-Irwin S M; Donovan F M; Cunningham D D; Van Nostrand W E
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, College of Medicine, University of California, Irvine 92717-4025.
CONTRACT NUMBER: AG00538 (NIA)
AG0096-11 (NIA)
HL49566 (NHLBI)
+
SOURCE: The Journal of biological chemistry, (1994 Sep 9) Vol. 269, No. 36, pp. 22623-7.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 13 Oct 1994
Last Updated on STN: 3 Feb 1997
Entered Medline: 4 Oct 1994

AB The amyloid beta-protein (A beta) and protease nexin-2/amyloid beta- protein precursor (PN-2/A beta PP) are major constituents of senile plaques and cerebrovascular deposits in individuals with Alzheimer's disease and related disorders. It has been suggested that the coagulation protease thrombin may process A beta PP in a manner leading to the formation of A beta. Here we investigated the effects of thrombin on the secretion and processing of PN-2/A beta PP and the production of A beta in a cellular system. Incubation of glioblastoma cells with thrombin (1-5 nM) resulted in the accumulation of abnormally processed, carboxyl-terminal-truncated forms of secreted PN-2/A beta PP (approximately 85 kDa) in the culture medium. Higher concentrations of thrombin (> 10 nM) also increased the levels of secreted PN-2/A beta PP in cultured untransfected glioblastoma cells and glioblastoma cells that were stably transfected to overproduce the 695 isoform of A beta PP. Increased secretion of PN-2/A beta PP required the proteolytic activity of thrombin, was induced by activation of the thrombin receptor by agonist peptides, and required activation of protein kinase C. Incubation of the untransfected and transfected glioblastoma cells with thrombin led to decreased levels of soluble A beta in the culture medium consistent with previously suggested mechanisms regarding the secretion of PN-2/A beta PP. Although the present studies suggest that thrombin does not directly contribute to A beta formation, its proteolysis of secreted PN-2/A beta PP may disrupt regions near the carboxyl terminus of the secreted proteins that account for their neuroprotective and cell adhesive properties.

TI Thrombin receptor activation induces secretion and nonamyloidogenic

processing of amyloid beta-protein precursor.
SO The Journal of biological chemistry, (1994 Sep 9) Vol. 269, No. 36, pp. 22623-7.
Journal code: 2985121R. ISSN: 0021-9258.
AB The amyloid beta-protein (A beta) and protease nexin-2/amyloid beta- protein precursor (PN-2/A beta PP) are major constituents of senile plaques and cerebrovascular deposits in individuals with Alzheimer's disease and related disorders. It has been suggested that the coagulation protease thrombin may process A beta PP in a manner leading to the formation of A beta. Here we investigated the effects of thrombin on the secretion and processing of PN-2/A beta PP and the production of A beta in a cellular system. Incubation of glioblastoma cells with thrombin (1-5 nM) resulted in the accumulation of abnormally processed, carboxyl-terminal-truncated forms of secreted PN-2/A beta PP (approximately 85 kDa) in the culture medium. Higher concentrations of thrombin (> 10 nM) also increased the levels of secreted PN-2/A beta PP in cultured untransfected glioblastoma cells and glioblastoma cells that were stably transfected to overproduce the 695 isoform of A beta PP. Increased secretion of PN-2/A beta PP required the proteolytic activity of thrombin, was induced by activation of the thrombin receptor by agonist peptides, and required activation of protein kinase C. Incubation of the untransfected and transfected glioblastoma cells with thrombin led to decreased levels of soluble A beta in the culture medium consistent with previously suggested mechanisms regarding the secretion of PN-2/A beta PP. Although the present studies suggest that thrombin does not directly contribute to A beta formation, its proteolysis of secreted PN-2/A beta PP may disrupt regions near the carboxyl terminus of the secreted proteins that account for their neuroprotective and cell adhesive properties.
CT Amyloid beta-Protein Precursor: BI, biosynthesis
*Amyloid beta-Protein Precursor: ME, metabolism
Cell Line
Dose-Response Relationship, Drug
Glioblastoma
Humans
Immunoblotting
Kinetics
Protein Kinase C: ME, metabolism
*Protein Processing, Post-Translational
Receptors, Thrombin: DE, drug effects
*Receptors, Thrombin: PH, physiology
*Thrombin: PD, pharmacology
Transfection
Tumor Cells, Cultured
CN 0 (Amyloid beta-Protein Precursor); 0 (Receptors, Thrombin); EC 2.7.1.37 (Protein Kinase C); EC 3.4.21.5 (Thrombin)

L152 ANSWER 27 OF 36 MEDLINE on STN
ACCESSION NUMBER: 2005395151 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16054018
TITLE: Understanding molecular mechanisms of proteolysis in Alzheimer's disease: progress toward therapeutic interventions.
AUTHOR: Higuchi Makoto; Iwata Nobuhisa; Saido Takaomi C
CORPORATE SOURCE: Laboratory for Proteolytic Neuroscience, RIKEN Brain Science Institute, 2-1 Hirosawa, Wako, Saitama 351-0198, Japan.. mhiguchi@brain.riken.jp
SOURCE: Biochimica et biophysica acta, (2005 Aug 1) Vol. 1751, No. 1, pp. 60-7. Electronic Publication: 2005-03-17. Ref: 61
Journal code: 0217513. ISSN: 0006-3002.

PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 2 Aug 2005
Last Updated on STN: 28 Sep 2005
Entered Medline: 27 Sep 2005
AB Amyloid beta peptide (Abeta) is not only a major constituent of extracellular fibrillary pathologies in Alzheimer's disease (AD) brains, but is also physiologically produced and metabolized in neurons. This fact led us to the notion that an age-related decrease in Abeta catabolism may contribute to the molecular pathogenesis of AD, providing a rationale for seeking proteolytic enzymes that degrade Abeta in the brain. Our recent studies have demonstrated that neprilysin is the most potent Abeta-degrading enzyme in vivo. Deficiency of endogenous neprilysin elevates the level of Abeta in brains of neprilysin-knockout mice in a gene dose-dependent manner, and an age-associated decline of neprilysin occurs in several regions of mouse brain. Neuropathological alterations in these same regions have been implicated in cognitive impairments of AD patients at an early stage of the disease. Furthermore, the level of neprilysin mRNA has been found to be significantly and selectively reduced in the hippocampus and temporal cortex of AD patients. A clarification of the role played by decreased neprilysin activity in the pathogenesis of AD has opened up the possibility of neprilysin up-regulation as a novel preventive and therapeutic approach to AD. Since the expression level and activity of neprilysin are likely to be regulated by neuropeptides and their receptors, non-peptidic agonists for these receptors might be effective agents to maintain a sufficient level of Abeta catabolism in brains of the elderly. In addition to Abeta deposits, intraneuronal fibrillary lesions, such as neurofibrillary tangles, are also a pathological hallmark of AD, and the extent of the resultant cytoskeletal disruptions may be dependent upon the activity levels of proteolytic enzymes. Among proteases for which major cytoskeletal components are good substrates, calpains were shown to participate in excitotoxic stress-induced neuritic degeneration in our recent analysis using genetically engineered mice. Moreover, we have found that this pathology can be reduced by controlling the activity of an endogenous calpain inhibitor known as calpastatin, providing a possible approach for the treatment of diverse neurodegenerative disorders, including AD.
TI Understanding molecular mechanisms of proteolysis in Alzheimer's disease: progress toward therapeutic interventions.
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
AB Amyloid beta peptide (Abeta) is not only a major constituent of extracellular fibrillary pathologies in Alzheimer's disease (AD) brains, but is also physiologically produced and metabolized in neurons. This fact led us to the notion that an age-related decrease in Abeta catabolism may contribute to the molecular pathogenesis of AD, providing a rationale for seeking proteolytic enzymes that degrade Abeta in the brain. Our recent studies have demonstrated that neprilysin is the most potent Abeta-degrading enzyme in vivo. Deficiency of endogenous neprilysin elevates the level of Abeta in brains of neprilysin-knockout mice in a gene dose-dependent manner, and an age-associated decline of neprilysin occurs in several regions of mouse brain. Neuropathological alterations in these same regions have been implicated in cognitive impairments of AD patients at an early stage of the disease. Furthermore, the level of neprilysin mRNA has been found to be significantly and selectively reduced in the hippocampus and temporal cortex of AD patients. A clarification of the role played by decreased neprilysin activity in the pathogenesis of AD has opened up the possibility of neprilysin up-regulation as a novel preventive and therapeutic approach to AD. Since the expression

level and activity of neprilysin are likely to be regulated by neuropeptides and their receptors, non-peptidic agonists for these receptors might be effective agents to maintain a sufficient level of Abeta catabolism in brains of the elderly. In addition to Abeta deposits, intraneuronal fibrillary lesions, such as neurofibrillary tangles, are also a pathological hallmark of AD, and the extent of the resultant cytoskeletal disruptions may be dependent upon the activity levels of proteolytic enzymes. Among proteases for which major cytoskeletal components are good substrates, calpains were shown to participate in excitotoxic stress-induced neuritic degeneration in our recent analysis using genetically engineered mice. Moreover, we have found that this pathology can be reduced by controlling the activity of an endogenous calpain inhibitor known as calpastatin, providing a possible approach for the treatment of diverse neurodegenerative disorders, including AD.

CT Alzheimer Disease: DT, drug therapy
Alzheimer Disease: PA, pathology
*Alzheimer Disease: PP, physiopathology
Amyloid Precursor Protein Secretases
*Amyloid beta-Protein: ME, metabolism
Amyloid beta-Protein Precursor: ME, metabolism
Animals
Aspartic Endopeptidases: ME, metabolism
Brain: EN, enzymology
Calcium-Binding Proteins: ME, metabolism
Calpain: AI, antagonists & inhibitors
Calpain: ME, metabolism
Cysteine Proteinase Inhibitors: TU, therapeutic use
Endopeptidases
Humans
Neprilysin: BI, biosynthesis
*Neprilysin: ME, metabolism
Neurites: PH, physiology
Up-Regulation
tau Proteins: ME, metabolism

CN 0 (Amyloid beta-Protein); 0 (Amyloid beta-Protein Precursor); 0 (Calcium-Binding Proteins); 0 (Cysteine Proteinase Inhibitors); 0 (tau Proteins); EC 3.4.- (Amyloid Precursor Protein Secretases); EC 3.4.- (Endopeptidases); EC 3.4.22.- (Calpain); EC 3.4.23.- (Aspartic Endopeptidases); EC 3.4.23.46 (BACE1 protein, human); EC 3.4.23.46 (Bace1 protein, mouse); EC 3.4.24.11 (Neprilysin)

L152 ANSWER 28 OF 36 MEDLINE on STN
ACCESSION NUMBER: 2004600336 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 15573708
TITLE: [Effects of central administration of beta-amyloid peptide (25-35): pathomorphological changes in the hippocampus and impairments of spatial memory]. Issledovanie effektov tsentral'nogo vvedeniia beta-amiloidnogo peptida (25-35): patomorfologicheskie izmeneniia v gippokampe i narushenie prostranstvennoi pamiati.
AUTHOR: Stepanichev M Iu; Zdobnova I M; Zarubenko I I; Lazareva N A; Guliaeva N V
SOURCE: Zhurnal vysshei nervnoi deiatelnosti imeni I P Pavlova, (2004 Sep-Oct) Vol. 54, No. 5, pp. 705-11. Journal code: 9421551. ISSN: 0044-4677.
PUB. COUNTRY: Russia: Russian Federation
DOCUMENT TYPE: (ENGLISH ABSTRACT) Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200503
ENTRY DATE: Entered STN: 3 Dec 2004
Last Updated on STN: 22 Mar 2005
Entered Medline: 21 Mar 2005

AB A possible relationship between the amnesia induced by central administration of beta-amyloid (25-35) [Abeta(25-35)] and neurodegeneration in the hippocampus was studied. Male Wistar rats received a single intracerebroventricular injection of Abeta(25-35) at a dose of 15 nmol. One month after the administration, animals were trained in an eight-arm radial maze. After the training, a histopathological investigation of the hippocampus was carried out using brain slices stained with hematoxylin/eosin. Abeta(25-35) induced impairments in reference and working memory in the eight-arm radial maze. A moderate decrease in neuronal cell number was demonstrated in the CA1, but not in the CA3.subfield of the hippocampus. The number of both reference and working errors negatively correlated with the number of neurons in hippocampal CA1. The results are the first evidence for a specific relationship between neurodegeneration in the CA1 subfield of rat hippocampus and impairments of learning and memory induced by Abeta(25-35).

TI [Effects of central administration of beta-amyloid peptide (25-35): pathomorphological changes in the hippocampus and impairments of spatial memory]. Issledovanie effektov tsentral'nogo vvedeniia beta-amiloidnogo peptida (25-35): patomorfologicheskie izmeneniia v gippokampe i narushenie prostranstvennoi pamiati.

SO Zhurnal vysshei nervnoi deiatelnosti imeni I P Pavlova, (2004 Sep-Oct) Vol. 54, No. 5, pp. 705-11. Journal code: 9421551. ISSN: 0044-4677.

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CT Check Tags: Male
Amyloid beta-Protein: AD, administration & dosage
*Amyloid beta-Protein: PD, pharmacology
Animals
Hippocampus: DE, drug effects
*Hippocampus: PA, pathology
Hippocampus: PP, physiopathology
Injections, Intraventricular
Maze Learning: DE, drug effects
*Maze Learning: PH, physiology
Memory: DE, drug effects
*Memory: PH, physiology
Memory Disorders: ET, etiology
Memory Disorders: PA, pathology
Memory Disorders: PP, physiopathology
Neurons: PA, pathology
Neurons: PH, physiology

Peptide Fragments: AD, administration & dosage
*Peptide Fragments: PD, pharmacology
Rats
Rats, Wistar
Space Perception: DE, drug effects
*Space Perception: PH, physiology
CN 0 (Amyloid beta-Protein); 0 (Peptide Fragments); 0 (amyloid beta-protein (25-35))

L152 ANSWER 29 OF 36 MEDLINE on STN
ACCESSION NUMBER: 2004083968 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 14973420
TITLE: Non-oncologic applications of radiolabeled peptides in nuclear medicine.
AUTHOR: Knight L C
CORPORATE SOURCE: Nuclear Medicine Division, Department of Diagnostic Imaging, Temple University School of Medicine, University Hospital, 3401 N. Broad Street, Philadelphia, PA 19140, USA.. lknight@temple.edu
CONTRACT NUMBER: R01 CA 96792 (NCI)
R01 HL 54578 (NHLBI)
SOURCE: The quarterly journal of nuclear medicine : official publication of the Italian Association of Nuclear Medicine (AIMN) [and] the International Association of Radiopharmacology (IAR), (2003 Dec) Vol. 47, No. 4, pp. 279-91. Ref: 58
Journal code: 9512274. ISSN: 1125-0135.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200405
ENTRY DATE: Entered STN: 20 Feb 2004
Last Updated on STN: 20 May 2004
Entered Medline: 19 May 2004

AB Radiolabeled peptides have been investigated for diagnostic imaging in a variety of non-oncologic diseases. For imaging thromboembolic disease, peptides which bind to various components of thrombi have been tested. For targeting the fibrin component of thrombi, peptide analogues of fibrin or fragments of fibronectin which have a distinct binding domain for fibrin have been studied. For targeting activated platelets within thrombi, linear and cyclic peptide antagonists of the glycoprotein IIb/IIIa receptor on platelets have been studied, as well as naturally occurring antagonists of this receptor which are found in venoms. Analogues of laminin and thrombospondin which bind to other receptors on platelets have also been tested. There is an approach which uses a peptide to target thrombin which is sequestered within a fibrin clot. Another area of investigation has been to develop an improved radiopharmaceutical for imaging sites of infection and/or inflammation. Peptides which would bind to leukocytes in vivo, such as antagonists to the tuftsin receptor, chemotactic peptides, interleukin-8, or a platelet factor 4 analogue, have been radiolabeled for this purpose. These agents would enable imaging of both infection and inflammation. Development of a radiopharmaceutical for specifically imaging infection has focused on antimicrobial peptides such as human neutrophil defensin, ubiquicidin, human lactoferrin and alafosfalin, which are expected to bind selectively to microorganisms and not to leukocytes. Radiolabeled peptides are also being explored as agents for assessing unstable atherosclerotic plaque (endothelin),

amyloid deposits (amyloid beta peptides), and the consequences of diabetes mellitus (human C-peptide).
TI Non-oncologic applications of radiolabeled peptides in nuclear medicine.
SO The quarterly journal of nuclear medicine : official publication of the Italian Association of Nuclear Medicine (AIMN) [and] the International Association of Radiopharmacology (IAR), (2003 Dec) Vol. 47, No. 4, pp. 279-91. Ref: 58
Journal code: 9512274. ISSN: 1125-0135.
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)
AB Radiolabeled peptides have been investigated for diagnostic imaging in a variety of non-oncologic diseases. For imaging thromboembolic disease, peptides which bind to various components of thrombi have been tested. For targeting the fibrin component of thrombi, peptide analogues of fibrin or fragments of fibronectin which have a distinct binding domain for fibrin have been studied. For targeting activated platelets within thrombi, linear and cyclic peptide antagonists of the glycoprotein IIb/IIIa receptor on platelets have been studied, as well as naturally occurring antagonists of this receptor which are found in venoms. Analogues of laminin and thrombospondin which bind to other receptors on platelets have also been tested. There is an approach which uses a peptide to target thrombin which is sequestered within a fibrin clot. Another area of investigation has been to develop an improved radiopharmaceutical for imaging sites of infection and/or inflammation. Peptides which would bind to leukocytes in vivo, such as antagonists to the tuftsin receptor, chemotactic peptides, interleukin-8, or a platelet factor 4 analogue, have been radiolabeled for this purpose. These agents would enable imaging of both infection and inflammation. Development of a radiopharmaceutical for specifically imaging infection has focused on antimicrobial peptides such as human neutrophil defensin, ubiquicidin, human lactoferrin and alafosfalin, which are expected to bind selectively to microorganisms and not to leukocytes. Radiolabeled peptides are also being explored as agents for assessing unstable atherosclerotic plaque (endothelin), amyloid deposits (amyloid beta peptides), and the consequences of diabetes mellitus (human C-peptide).
CT *Alzheimer Disease: RI, radionuclide imaging
Antimicrobial Cationic Peptides: DU, diagnostic use
*Arteriosclerosis: RI, radionuclide imaging
*Diabetes Insipidus: RI, radionuclide imaging
Humans
*Infection: RI, radionuclide imaging
*Inflammation: RI, radionuclide imaging
Neoplasms: RI, radionuclide imaging
Nuclear Medicine: MT, methods
*Peptides: DU, diagnostic use
*Radiopharmaceuticals: DU, diagnostic use
*Thrombosis: RI, radionuclide imaging

L152 ANSWER 30 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 5
ACCESSION NUMBER: 2002:136047 BIOSIS Full-text
DOCUMENT NUMBER: PREV200200136047
TITLE: Receptors for chemotactic formyl peptides as pharmacological targets.
AUTHOR(S): Le, Yingying [Reprint author]; Yang, Yiming; Cui, Youhong; Yazawa, Hiroshi; Gong, Wanghua; Qiu, Cunping; Wang, Ji Ming
CORPORATE SOURCE: Laboratory of Molecular Immunoregulation, Center for Cancer Research, National Cancer Institute at Frederick, Frederick, MD, 21702, USA

ley@mail.ncifrf.gov
SOURCE: International Immunopharmacology, (January, 2002) Vol. 2, No. 1, pp. 1-13. print.
ISSN: 1567-5769.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Feb 2002
Last Updated on STN: 26 Feb 2002

AB Leukocytes accumulate at sites of inflammation and immunological reaction in response to locally existing chemotactic mediators. N-formyl peptides, such as fMet-Leu-Phe (fMLF), are some of the first identified and most potent chemoattractants for phagocytic leukocytes. In addition to the bacterial peptide fMLF and the putative endogenously produced formylated peptides, a number of novel peptide agonists have recently been identified that selectively activate the high-affinity fMLF receptor FPR and/or its low-affinity variant FPRL1, both of which belong to the seven-transmembrane (STM), G protein-coupled receptor (GPCR) superfamily. These agonists include peptide domains derived from the envelope proteins of human immunodeficiency virus type 1 (HIV-1) and at least three amyloidogenic polypeptides, the human acute phase protein serum amyloid A, the 42 amino acid form of beta amyloid peptide and a 21 amino acid fragment of human prion. Furthermore, a cleavage fragment of neutrophil granule-derived bactericidal cathelicidin, LL-37, is also a chemotactic agonist for FPRL1. Activation of formyl peptide receptors results in increased cell migration, phagocytosis, release of proinflammatory mediators, and the signaling cascade culminates in heterologous desensitization of other STM receptors including chemokine receptors CCR5 and CXCR4, two coreceptors for HIV-1. Thus, by interacting with a variety of exogenous and host-derived agonists, formyl peptide receptors may play important roles in proinflammatory and immunological diseases and constitute a novel group of pharmacological targets.

TI Receptors for chemotactic formyl peptides as pharmacological targets.

SO International Immunopharmacology, (January, 2002) Vol. 2, No. 1, pp. 1-13. print.
ISSN: 1567-5769.

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IT Major Concepts
Biochemistry and Molecular Biophysics; Immune System (Chemical

Coordination and Homeostasis); Pharmacology
IT Parts, Structures, & Systems of Organisms
leukocytes: blood and lymphatics, immune system
IT Diseases
Alzheimer's disease: behavioral and mental disorders, nervous system disease
Alzheimer Disease (MeSH)
IT Diseases
immunological diseases: immune system disease
IT Diseases
prion diseases: prion disease
Prion Diseases (MeSH)
IT Diseases
proinflammatory diseases: immune system disease
IT Chemicals & Biochemicals
G-protein-coupled receptor; chemotactic formyl peptide receptors: pharmacological targets; chemotactic formyl peptides; fMet-Leu-Phe; non-steroidal antiinflammatory drugs

L152 ANSWER 31 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:205264 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400205791
TITLE: Possible role of Nogo - A in Alzheimer 's disease: association with Abeta plaques.
AUTHOR(S): Prinjha, R. K. [Reprint Author]; Hussain, I. [Reprint Author]; Kumar, U. [Reprint Author]; Richardson, J. C. [Reprint Author]; Harper, A. J. [Reprint Author]; Vinson, M. [Reprint Author]; Burbidge, S. A. [Reprint Author]; Parsons, A. A. [Reprint Author]; Howlett, D. [Reprint Author]
CORPORATE SOURCE: Alzheimer's Dis. Res., Neurol.-GI CEDD, GlaxoSmithKline, Harlow, UK
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 880.3. http://sfn.scholarone.com. e-file.
Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

AB The function of axonal outgrowth inhibitors such as Nogo-A in blocking regeneration after CNS trauma is well known. We have previously described alterations in expression and function of Nogo-A in ALS (DuPuis et al 2002) and ischaemic stroke (Roberts et al 2002) but its possible role in the mechanisms of Alzheimer's disease remains poorly understood. Transgenic mice overexpressing the human APPswedish mutant protein have been shown, on ageing, to deposit plaques composed of Abeta peptide. Immunohistochemical analysis in this model, has identified abundant amyloid plaques in the cortex and hippocampus. The distribution of a wide range of cell-type markers including GFAP and neurofilament has been employed in sections from these animals. Of all these markers only Nogo-A and BACE (the enzyme responsible for amyloid peptide production from APP) were found to display immunoreactivity in a structure forming a halo around the amyloid plaque. Temporal studies from 12 months onwards suggest that the Abeta plaques begin as small focal deposits that grow outwards. The pattern of Nogo-A and BACE staining at the periphery of plaques suggest that they may have a role in liberating soluble Abeta that

adds to the growing plaque. In SHSY5Y cells expressing mutant (swedish) APP, both transfected Nogo-A and B show prominent co-localisation with transfected BACE within the ER, a major site of Abeta production. A range of in vitro and in vivo models have been used to investigate this novel and intriguing interaction in more detail. In addition to its central role in blocking CNS regeneration our findings suggest a potentially important role for Nogo in the development of Alzheimer 's disease.

TI Possible role of Nogo - A in Alzheimer's disease: association with Abeta plaques.

SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 880.3. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.

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IT Major Concepts
Cardiovascular Medicine (Human Medicine, Medical Sciences); Nervous System (Neural Coordination); Neurology (Human Medicine, Medical Sciences); Psychiatry (Human Medicine , Medical Sciences)

IT Parts, Structures, & Systems of Organisms
CNS: nervous system; amyloid plaques: nervous system; hippocampus: nervous system; neurofilaments: nervous system; plaques: nervous system

IT Diseases
Alzheimer's disease: behavioral and mental disorders, nervous system disease
Alzheimer Disease (MeSH)

IT Diseases
central nervous system trauma: injury, nervous system disease

IT Diseases
stroke: nervous system disease, vascular disease
Cerebrovascular Disorders (MeSH)

IT Chemicals & Biochemicals
Abeta peptide; BACE; GFAP; Nogo; Nogo-A; amyloid; amyloid peptide

L152 ANSWER 32 OF 36 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

ACCESSION NUMBER: 2001:575419 BIOSIS Full-text
DOCUMENT NUMBER: PREV200100575419
TITLE: Enhanced antidepressant effect of sigma1 (sigma1) agonists in beta-amyloid peptide-treated rodents.
AUTHOR(S): Urani, A. [Reprint author]; Romieu, P.; Roman, F. J. [Reprint author]; Noda, Y.; Kamei, H.; Tran, M. H.; Nagai, T.; Nabeshima, T.; Maurice, T.
CORPORATE SOURCE: Biochimie/Enzymologie, Pfizer GRD, Fresnes, France
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 853. print. Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001. ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting) Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2001 Last Updated on STN: 25 Feb 2002

AB The sigma1 receptor is a 223 amino acide protein involved in numerous behavioral effects. In particular, sigma1 receptor agonists present potent antidepressant-like effects in several animal models of behavioral despair. The antidepressant efficacy of selective sigma1 agonists was studied in two models of beta-amyloid-induced cognitive deficits. First, in mice injected centrally with beta25-35-amyloid peptide and submitted ten days after to forced swim test. In this test, igmesine appeared more efficient in beta25-35 animals, by reducing immobility at 30 mg/kg vs. 60 mg/kg in control groups. Such facilitation was not observed with desipramine. Furthermore, beta25-35 animals exhibited decreased progesterone levels in the hippocampus (-47%). Second, in rats infused during 14 days with the beta1-40 amyloid peptide and submitted to the conditioned fear stress. In this test, (+)-SKF-10,047 reduced the stress-induced motor suppression at 3 mg/kg in beta1-40 peptide infused rats, vs. 6 mg/kg in beta40-1 treated rats. Igmesine presented an effect at 10 mg/kg in beta1-40 infused rats vs. 30 mg/kg in control rats. Neurosteroid measurements and immunohistochemical studies will also be presented. The sigma1 agonist efficacy is known to depend on neuro(active)steroids levels, synthesized mainly by glial cells. These cells may be affected by b-amyloid toxicity. We suggest that sigma1 agonists, due to their enhanced efficacy, may improve Alzheimer 's disease-related cognitive deficits.

TI Enhanced antidepressant effect of sigma1 (sigma1) agonists in beta-amyloid peptide-treated rodents.

SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 853. print. Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001. ISSN: 0190-5295.

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IT Major Concepts
Behavior; Nervous System (Neural Coordination); Pharmacology

IT Parts, Structures, & Systems of Organisms
glial cell: nervous system; hippocampus: nervous system

IT Diseases
Alzheimer's disease: behavioral and mental disorders, nervous system disease
Alzheimer Disease (MeSH)

IT Diseases
cognitive deficit: behavioral and mental disorders, nervous system disease
Cognition Disorders (MeSH)

IT Chemicals & Biochemicals
SKF-10,047; beta-amyloid 1-40 peptide: central administration, toxicity; beta-amyloid 25-35 peptide: central administration, toxicity; desipramine: antidepressant-drug, pharmacodynamics, potency, sigma-1 agonist; igmesine: antidepressant-drug, pharmacodynamics, potency, sigma-1 agonist; progesterone: regulation

L152 ANSWER 33 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2007455920 EMBASE Full-text
TITLE: DNA vaccine and the CNS axonal regeneration.
AUTHOR: Nie D.-Y.; Xu G.; Ahmed S.; Xiao Z.-C.
CORPORATE SOURCE: Z.-C. Xiao, Department of Clinical Research, Singapore General Hospital, Singapore, Singapore. xiao.zhi.cheng@sgh.com.sg
SOURCE: Current Pharmaceutical Design, (Aug 2007) Vol. 13, No. 24, pp. 2500-2506.
Refs: 110
ISSN: 1381-6128 CODEN: CPDEFP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
032 Psychiatry
036 Health Policy, Economics and Management
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Oct 2007
Last Updated on STN: 17 Oct 2007

AB Vaccines have been considered in treating many CNS degenerative disorders, including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), epilepsy, multiple sclerosis (MS), spinal cord injury (SCI), and stroke. DNA vaccines have emerged as novel therapeutic agents because of the

simplicity of their generation and application. Myelin domponents such as NOGO, MAG and OMGP are known to trigger demyelinating autoimmunity and to prevent axonal regeneration. For these reasons DNA vaccines encoding NOGO, MAG and OMGP, and fragments thereof, make them suitable vehicles for treatment of SCIs and MS. We need to obtain a deeper understanding of the immunologic mechanisms underlying the neuroprotective immunity to optimize the design of DNA vaccines for their use in clinical setting. In this review, we discuss recent findings suggesting that DNA vaccines hold a promising future for the treatment of axonal degeneration and demyelination. .COPYRGT. 2007 Bentham Science Publishers Ltd.

DT Journal; General Review; (Review)

AB Vaccines have been considered in treating many CNS degenerative disorders, including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), epilepsy, multiple sclerosis (MS), spinal cord injury (SCI), and stroke. DNA vaccines have emerged as novel therapeutic agents because of the simplicity of their generation and application. Myelin domponents such as NOGO, MAG and OMGP are known to trigger demyelinating autoimmunity and to prevent axonal regeneration. For these reasons DNA vaccines encoding NOGO, MAG and OMGP, and fragments thereof, make them suitable vehicles for treatment of SCIs and MS. We need to obtain a deeper understanding of the immunologic mechanisms underlying the neuroprotective immunity to optimize the design of DNA vaccines for their use in clinical setting. In this review, we discuss recent findings suggesting that DNA vaccines hold a promising future for the treatment of axonal degeneration and demyelination. .COPYRGT. 2007 Bentham Science Publishers Ltd.

CT Medical Descriptors:
Alzheimer disease: DT, drug therapy
Alzheimer disease: PC, prevention
autoimmunity
brain injury: DT, drug therapy
cost effectiveness analysis
*degenerative disease: DM, disease management
*degenerative disease: DT, drug therapy
*degenerative disease: PC, prevention
demyelination
drug cost
drug efficacy
drug safety
drug targeting
epilepsy: DT, drug therapy
epilepsy: PC, prevention
human
humoral immunity
Huntington chorea: DT, drug therapy
Huntington chorea: PC, prevention
immunization
multiple sclerosis: DT, drug therapy
multiple sclerosis: PC, prevention
*nerve fiber regeneration
neuroprotection
nonhuman
Parkinson disease: DT, drug therapy
Parkinson disease: PC, prevention
priority journal
review
spinal cord injury: DT, drug therapy
spinal cord injury: PC, prevention
stroke: DT, drug therapy
stroke: PC, prevention
T lymphocyte activation

CT Drug Descriptors:
Alzheimer disease vaccine: DT, drug therapy
amyloid beta protein: DT, drug therapy
brevican: EC, endogenous compound
chondroitin ABC lyase: DT, drug therapy
chondroitin ABC lyase: PD, pharmacology
dendritic cell vaccine: DT, drug therapy
*DNA vaccine: AD, drug administration
*DNA vaccine: DT, drug therapy
*DNA vaccine: LY, intralymphatic drug administration
*DNA vaccine: IM, intramuscular drug administration
*DNA vaccine: NA, intranasal drug administration
*DNA vaccine: PO, oral drug administration
*DNA vaccine: PE, pharmacoeconomics
in 1: DT, drug therapy
in 1: PD, pharmacology
matrix metalloproteinase: DT, drug therapy
matrix metalloproteinase: PD, pharmacology
monoclonal antibody: DT, drug therapy
monoclonal antibody: PD, pharmacology
myelin associated glycoprotein: EC, endogenous compound
myelin basic protein: DT, drug therapy
neurocan: EC, endogenous compound
neuromodulin: EC, endogenous compound
Nogo 66 receptor: EC, endogenous compound
protein Nogo: EC, endogenous compound
protein p75: EC, endogenous compound
proteochondroitin sulfate: EC, endogenous compound
recombinant vaccine: DT, drug therapy
tenascin: EC, endogenous compound
versican: EC, endogenous compound
RN (amyloid beta protein) 109770-29-8; (chondroitin ABC lyase) 9024-13-9; (neurocan) 170276-50-3; (protein p75) 91608-97-8; (versican) 126968-45-4

L152 ANSWER 34 OF 36 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2007455917 EMBASE Full-text
TITLE: Targeting the Nogo-a signalling pathway to promote recovery following acute CNS injury.
AUTHOR: Walmsley A.R.; Mir A.K.
CORPORATE SOURCE: A.R. Walmsley, Novartis Institutes for Biomedical Research, 4056 Basel, Switzerland. andrian_robert.walmsley@novartis.com
SOURCE: Current Pharmaceutical Design, (Aug 2007) Vol. 13, No. 24, pp. 2470-2484.
Refs: 151
ISSN: 1381-6128 CODEN: CPDEFP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Oct 2007
Last Updated on STN: 17 Oct 2007
AB Functional recovery following acute CNS injury in humans, such as spinal cord injury and stroke, is exceptionally limited, leaving the affected individual

with life-long neurological deficits such as loss of limb movement and sensation leading to a compromised quality of life. As yet, there is no effective treatment on the market for such injuries. This lack of functional recovery can at least in part be attributed to the restriction of axonal regeneration and neuroplasticity by several CNS myelin proteins that have been shown to be potent inhibitors of neurite outgrowth in vitro, namely myelin-associated glycoprotein (MAG), Nogo-A and oligodendrocyte myelin glycoprotein (OMgp). Nogo-A contains multiple neurite outgrowth inhibitory domains exposed on the surface of myelinating oligodendrocytes located within its amino-terminal region (amino-Nogo-A) and C-terminal region (Nogo-66). Although structurally dissimilar; Nogo-66, MAG and OMgp exert their inhibitory effects by binding the GPI-linked neuronal Nogo-66 receptor (NgR) that transduces the inhibitory signal to the cell interior via transmembrane co-receptors LINGO-1 and p75(NTR) or TROY. Although the receptor(s) for amino-Nogo-A are unknown, amino-Nogo-A and NgR ligands mutually activate the small GTPase RhoA. Consistent with their neurite outgrowth inhibitory function, approaches counter-acting Nogo-A using function-blocking antibodies, NgR using peptide antagonists and receptor bodies or RhoA using deactivating enzymes have been shown to significantly enhance axonal regeneration and neuroplasticity leading to improved functional recovery in animal models of acute CNS injury. These in vivo findings thus provide a sound basis for the development of an effective treatment for acute CNS injuries in humans. .COPYRGT. 2007 Bentham Science Publishers Ltd.
DT Journal; General Review; (Review)
AB Functional recovery following acute CNS injury in humans, such as spinal cord injury and stroke, is exceptionally limited, leaving the affected individual with life-long neurological deficits such as loss of limb movement and sensation leading to a compromised quality of life. As yet, there is no effective treatment on the market for such injuries. This lack of functional recovery can at least in part be attributed to the restriction of axonal regeneration and neuroplasticity by several CNS myelin proteins that have been shown to be potent inhibitors of neurite outgrowth in vitro, namely myelin-associated glycoprotein (MAG), Nogo-A and oligodendrocyte myelin glycoprotein (OMgp). Nogo-A contains multiple neurite outgrowth inhibitory domains exposed on the surface of myelinating oligodendrocytes located within its amino-terminal region (amino-Nogo-A) and C-terminal region (Nogo-66). Although structurally dissimilar; Nogo-66, MAG and OMgp exert their inhibitory effects by binding the GPI-linked neuronal Nogo-66 receptor (NgR) that transduces the inhibitory signal to the cell interior via transmembrane co-receptors LINGO-1 and p75(NTR) or TROY. Although the receptor(s) for amino-Nogo-A are unknown, amino-Nogo-A and NgR ligands mutually activate the small GTPase RhoA. Consistent with their neurite outgrowth inhibitory function, approaches counter-acting Nogo-A using function-blocking antibodies, NgR using peptide antagonists and receptor bodies or RhoA using deactivating enzymes have been shown to significantly enhance axonal regeneration and neuroplasticity leading to improved functional recovery in animal models of acute CNS injury. These in vivo findings thus provide a sound basis for the development of an effective treatment for acute CNS injuries in humans. .COPYRGT. 2007 Bentham Science Publishers Ltd.
CT Medical Descriptors:
amino terminal sequence
carboxy terminal sequence
*central nervous system disease: DT, drug therapy
continuous infusion
drug efficacy
drug receptor binding
drug targeting
enzyme activation
enzyme inhibition
human

nerve cell plasticity
nerve fiber growth
nerve fiber regeneration
neuroprotection
nonhuman
oligodendroglia
  optic nerve injury: DT, drug therapy
priority journal
protein domain
quality of life
review
signal transduction
  spinal cord injury: DT, drug therapy
stroke
CT Drug Descriptors:
  amyloid beta protein: EC, endogenous compound
  amyloid precursor protein: EC, endogenous compound
beta secretase: EC, endogenous compound
bucladesine: CB, drug combination
  bucladesine: DT, drug therapy
  bucladesine: PD, pharmacology
calcium: EC, endogenous compound
cyclic AMP: EC, endogenous compound
cyclic AMP dependent protein kinase: EC, endogenous compound
cyclic AMP responsive element binding protein: EC, endogenous compound
  epidermal growth factor receptor kinase inhibitor: DT, drug therapy
  epidermal growth factor receptor kinase inhibitor: PD, pharmacology
immunoglobulin G1 antibody: CM, drug comparison
  immunoglobulin G1 antibody: DT, drug therapy
  immunoglobulin G1 antibody: PD, pharmacology
  in 1: DT, drug therapy
  in 1: CE, intracerebral drug administration
  in 1: PD, pharmacology
mitogen activated protein kinase: EC, endogenous compound
monoclonal antibody: CM, drug comparison
  monoclonal antibody: DT, drug therapy
  monoclonal antibody: PD, pharmacology
  monoclonal antibody 11C7: DT, drug therapy
  monoclonal antibody 11C7: TL, intrathecal drug administration
  monoclonal antibody 11C7: PD, pharmacology
monoclonal antibody 7B12: CM, drug comparison
  monoclonal antibody 7B12: DT, drug therapy
  monoclonal antibody 7B12: CV, intracerebroventricular drug administration
  monoclonal antibody 7B12: TL, intrathecal drug administration
  monoclonal antibody 7B12: PD, pharmacology
myelin associated glycoprotein: EC, endogenous compound
myelin protein: EC, endogenous compound
neurotrophin: EC, endogenous compound
  Nogo 66 receptor: EC, endogenous compound
phosphodiesterase IV: EC, endogenous compound
protein kinase C: EC, endogenous compound
  protein kinase C inhibitor: DT, drug therapy
  protein kinase C inhibitor: TL, intrathecal drug administration
  protein kinase C inhibitor: PD, pharmacology
*protein Nogo A: EC, endogenous compound
protein p75: EC, endogenous compound
RhoA guanine nucleotide binding protein: EC, endogenous compound

rolipram: CB, drug combination
  rolipram: DT, drug therapy
  rolipram: PD, pharmacology
  rolipram: SC, subcutaneous drug administration
tissue inhibitor of metalloproteinase 2: EC, endogenous compound
tissue inhibitor of metalloproteinase 3: EC, endogenous compound
tumor necrosis factor: EC, endogenous compound
unindexed drug
RN (amyloid beta protein) 109770-29-8; (bucladesine) 16980-89-5, 362-74-3; (calcium) 7440-70-2; (cyclic AMP responsive element binding protein) 130428-87-4, 130939-96-7; (cyclic AMP) 60-92-4; (mitogen activated protein kinase) 142243-02-5; (protein kinase C) 141436-78-4; (protein p75) 91608-97-8; (rolipram) 61413-54-5; (tissue inhibitor of metalloproteinase 2) 124861-55-8; (tissue inhibitor of metalloproteinase 3) 145809-21-8, 164781-40-2

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ACCESSION NUMBER: 2005306458 EMBASE Full-text
TITLE: NF-κB factor c-Rel mediates neuroprotection elicited by mGlu5 receptor agonists againts amyloid β-peptide toxicity.
AUTHOR: Pizzi M.; Sarnico I.; Boroni F.; Benarese M.; Steimberg N.; Mazzoleni G.; Dietz G.P.H.; Bahr M.; Liou H.-C.; Spano P.F.
CORPORATE SOURCE: M. Pizzi, Division of Pharmacology, Department of Biomedical Sciences and Biotechnologies, Viale Europa 11, 25123 Brescia, Italy. pizzi@med.unibs.it
SOURCE: Cell Death and Differentiation, (Jul 2005) Vol. 12, No. 7, pp. 761-772.
Refs: 89
ISSN: 1350-9047 CODEN: CDDIEK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jul 2005
Last Updated on STN: 28 Jul 2005

AB Opposite effects of nuclear factor-κB (NF-KB) on neuron survival rely on activation of diverse NF-κB factors. While p65 is necessary for glutamate-induced cell death, c-Rel mediates prosurvival effects of interleukin-1β. However, it is unknown whether activation of c-Rel-dependent pathways reduces neuron vulnerability to amyloid-β (Aβ), a peptide implicated in Alzheimer's disease pathogenesis. We show that neuroprotection elicited by activation of metabotropic glutamate receptors type 5 (mGlu5) against Aβ toxicity depends on c-Rel activation. A. beta. peptide induced NF-κB factors p50 and p65. The mGlu5 agonists activated c-Rel, besides p50 and p65, and the expression of manganese superoxide dismutase (MnSOD) and Bcl-X(L). Targeting c-Rel expression by RNA interference suppressed the induction of both antiapoptotic genes. Targeting c-Rel or Bcl-X(L) prevented the prosurvival effect of mGlu5 agonists. Conversely, c-Rel overexpression or TAT-Bcl-X(L) addition rescued neurons from Aβ toxicity. These data demonstrate that mGlu5 receptor activation promotes a c-Rel-dependent antiapoptotic pathway responsible for

neuroprotection against A. beta. peptide. .COPYRGT. 2005 Nature Publishing Group. All rights reserved.

TI NF-κB factor c-Rel mediates neuroprotection elicited by mGlu5 receptor agonists againts amyloid . beta.-peptide toxicity.

DT Journal; General Review; (Review)

AB Opposite effects of nuclear factor-κB (NF-KB) on neuron survival rely on activation of diverse NF-κB factors. While p65 is necessary for glutamate-induced cell death, c-Rel mediates prosurvival effects of interleukin-1β. However, it is unknown whether activation of c-Rel-dependent pathways reduces neuron vulnerability to amyloid-β (Aβ), a peptide implicated in Alzheimer's disease pathogenesis. We show that neuroprotection elicited by activation of metabotropic glutamate receptors type 5 (mGlu5) against Aβ toxicity depends on c-Rel activation. A. beta. peptide induced NF-κB factors p50 and p65. The mGlu5 agonists activated c-Rel, besides p50 and p65, and the expression of manganese superoxide dismutase (MnSOD) and Bcl-X(L). Targeting c-Rel expression by RNA interference suppressed the induction of both antiapoptotic genes. Targeting c-Rel or Bcl-X(L) prevented the prosurvival effect of mGlu5 agonists. Conversely, c-Rel overexpression or TAT-Bcl-X(L) addition rescued neurons from Aβ toxicity. These data demonstrate that mGlu5 receptor activation promotes a c-Rel-dependent antiapoptotic pathway responsible for neuroprotection against A. beta. peptide. .COPYRGT. 2005 Nature Publishing Group. All rights reserved.

CT Medical Descriptors:
*Alzheimer disease: ET, etiology
animal cell
cell survival
controlled study
drug mechanism
gene induction
gene overexpression
gene targeting
human
human cell
mouse
neuroprotection
neurotoxicity
nonhuman
pathogenesis
priority journal
protein expression
protein function
protein induction
protein targeting
review
RNA interference

CT Drug Descriptors:
2 chloro 5 hydroxyphenylglycine
3 hydroxyphenylglycine: PD, pharmacology
amino acid receptor stimulating agent: PD, pharmacology
*amyloid beta protein
*glutamate receptor 5: EC, endogenous compound
*glutamate receptor agonist: PD, pharmacology
immunoglobulin enhancer binding protein: EC, endogenous compound
protein bcl xl: EC, endogenous compound
protein p50: EC, endogenous compound
small interfering RNA: PD, pharmacology
superoxide dismutase: EC, endogenous compound
synaptotagmin: EC, endogenous compound

transactivator protein: EC, endogenous compound
*transcription factor Rel: EC, endogenous compound
unclassified drug

RN (amyloid beta protein) 109770-29-8; (protein bcl xl) 151033-38-4; (superoxide dismutase) 37294-21-6, 9016-01-7, 9054-89-1; (synaptotagmin) 134193-26-3, 134193-27-4

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ACCESSION NUMBER: 2005122738 EMBASE Full-text
TITLE: New therapeutic strategies and drug candidates for neurodegenerative diseases: p53 and TNF-α inhibitors, and GLP-1 receptor agonists
AUTHOR: Greig N.H.; Mattson M.P.; Perry T.; Chan S.L.; Giordano T.; Sambamurti K.; Rogers J.T.; Ovadia H.; Lahiri D.K.
CORPORATE SOURCE: N.H. Greig, Drug Design and Development Section, Laboratory of Neurosciences, Gerontology Research Center, 5600 Nathan Shock Drive, Baltimore, MD 21224, United States. greign@grc.nia.nih.gov
SOURCE: Annals of the New York Academy of Sciences, (2004) Vol. 1035, pp. 290-315.
Refs: 119
ISSN: 0077-8923 CODEN: ANYAA9
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
052 Toxicology
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 31 Mar 2005
Last Updated on STN: 31 Mar 2005

AB Owing to improving preventative, diagnostic, and therapeutic measures for cardiovascular disease and a variety of cancers, the average ages of North Americans and Europeans continue to rise. Regrettably, accompanying this increase in life span, there has been an increase in the number of individuals afflicted with age-related neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and stroke. Although different cell types and brain areas are vulnerable among these, each disorder likely develops from activation of a common final cascade of biochemical and cellular events that eventually lead to neuronal dysfunction and death. In this regard, different triggers, including oxidative damage to DNA, the overactivation of glutamate receptors, and disruption of cellular calcium homeostasis, albeit initiated by different genetic and/or environmental factors, can instigate a cascade of intracellular events that induce apoptosis. To forestall the neurodegenerative process, we have chosen specific targets to inhibit that are at pivotal rate-limiting steps within the pathological cascade. Such targets include TNF-α, p53, and GLP-1 receptor. The cytokine TNF-α is elevated in Alzheimer's disease, Parkinson's disease, stroke, and amyotrophic lateral sclerosis. Its synthesis can be reduced via posttranscriptional mechanisms with novel analogues of the classic drug, thalidomide. The intracellular protein and transcription factor, p53, is activated by the Alzheimer's disease toxic peptide, Aβ, as well as by excess glutamate and hypoxia to trigger neural cell death. It is inactivated by novel tetrahydrobenzothiazole and -oxazole analogues to rescue cells from lethal insults. Stimulation of the glucagon-like peptide-1 receptor (GLP-1R) in brain is associated with neurotrophic functions that, additionally, can protect cells against excess

glutamate and other toxic insults. .COPYRGT. 2004 New York Academy of Sciences.

TI New therapeutic strategies and drug candidates for neurodegenerative diseases: p53 and TNF-α inhibitors, and GLP-1 receptor agonists.

SO Annals of the New York Academy of Sciences, (2004) Vol. 1035, pp. 290-315.
Refs: 119
ISSN: 0077-8923 CODEN: ANYAA9

AB Owing to improving preventative, diagnostic, and therapeutic measures for cardiovascular disease and a variety of cancers, the average ages of North Americans and Europeans continue to rise. Regrettably, accompanying this increase in life span, there has been an increase in the number of individuals afflicted with age-related neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and stroke. Although different cell types and brain areas are vulnerable among these, each disorder likely develops from activation of a common final cascade of biochemical and cellular events that eventually lead to neuronal dysfunction and death. In this regard, different triggers, including oxidative damage to DNA, the overactivation of glutamate receptors, and disruption of cellular calcium homeostasis, albeit initiated by different genetic and/or environmental factors, can instigate a cascade of intracellular events that induce apoptosis. To forestall the neurodegenerative process, we have chosen specific targets to inhibit that are at pivotal rate-limiting steps within the pathological cascade. Such targets include TNF-α, p53, and GLP-1 receptor. The cytokine TNF-α is elevated in Alzheimer's disease, Parkinson's disease, stroke, and amyotrophic lateral sclerosis. Its synthesis can be reduced via posttranscriptional mechanisms with novel analogues of the classic drug, thalidomide. The intracellular protein and transcription factor, p53, is activated by the Alzheimer's disease toxic peptide, Aβ, as well as by excess glutamate and hypoxia to trigger neural cell death. It is inactivated by novel tetrahydrobenzothiazole and -oxazole analogues to rescue cells from lethal insults. Stimulation of the glucagon-like peptide-1 receptor (GLP-1R) in brain is associated with neurotrophic functions that, additionally, can protect cells against excess glutamate and other toxic insults. .COPYRGT. 2004 New York Academy of Sciences.

CT Medical Descriptors:
Alzheimer disease: DT, drug therapy
amyotrophic lateral sclerosis: DT, drug therapy
apoptosis
brain region
calcium homeostasis
cell type
conference paper
*degenerative disease: DT, drug therapy
drug synthesis
drug targeting
environmental factor
enzyme inhibition
genetic transcription
heredity
human
nerve cell necrosis
nonhuman
oxidative stress
Parkinson disease: DT, drug therapy
stroke: DT, drug therapy

CT Drug Descriptors:
2',6' dithiothalidomide: DV, drug development
2',6' dithiothalidomide: PD, pharmacology

3 thiothalidomide: DV, drug development
3 thiothalidomide: PD, pharmacology
3,2',6' trithiothalidomide: DV, drug development
3,2',6' trithiothalidomide: PD, pharmacology
3,6' dithiothalidomide: DV, drug development
3,6' dithiothalidomide: PD, pharmacology
6' thiothalidomide: DV, drug development
6' thiothalidomide: PD, pharmacology
amyloid beta protein: EC, endogenous compound
antiparasitic agent: PD, pharmacology
calcium: EC, endogenous compound
cyclooxygenase 1 inhibitor: DT, drug therapy
cyclooxygenase 1 inhibitor: PD, pharmacology
cyclooxygenase 2 inhibitor: DT, drug therapy
cyclooxygenase 2 inhibitor: PD, pharmacology
dithioglutarimide: DV, drug development
dithioglutarimide: PD, pharmacology
dithiophthalimide: DV, drug development
dithiophthalimide: PD, pharmacology
etanercept: IV, intravenous drug administration
etanercept: PD, pharmacology
etanercept: SC, subcutaneous drug administration
exendin 4: DV, drug development
exendin 4: DT, drug therapy
exendin 4: PD, pharmacology
glucagon like peptide 1: EC, endogenous compound
*glucagon like peptide 1 receptor agonist: DV, drug development
*glucagon like peptide 1 receptor agonist: DT, drug therapy
*glucagon like peptide 1 receptor agonist: PD, pharmacology
glutamate receptor: EC, endogenous compound
*hormone receptor stimulating agent: DV, drug development
*hormone receptor stimulating agent: DT, drug therapy
*hormone receptor stimulating agent: PD, pharmacology
infliximab: IV, intravenous drug administration
infliximab: PD, pharmacology
infliximab: SC, subcutaneous drug administration
oxazole derivative: DV, drug development
oxazole derivative: PD, pharmacology
pifithrin alpha: PD, pharmacology
*protein inhibitor: AN, drug analysis
*protein inhibitor: DV, drug development
*protein inhibitor: DT, drug therapy
*protein inhibitor: TO, drug toxicity
*protein inhibitor: PO, oral drug administration
*protein inhibitor: PD, pharmacology
protein p53: EC, endogenous compound
*protein p53 inhibitor: DV, drug development
*protein p53 inhibitor: DT, drug therapy
*protein p53 inhibitor: PD, pharmacology
tetrahydrobenzothiazole: DV, drug development
tetrahydrobenzothiazole: PD, pharmacology
thalidomide: AN, drug analysis
thalidomide: DV, drug development
thalidomide: DT, drug therapy
thalidomide: TO, drug toxicity
thalidomide: PO, oral drug administration
thalidomide: PD, pharmacology
thiazole derivative: DV, drug development
thiazole derivative: PD, pharmacology
tumor necrosis factor alpha: EC, endogenous compound

SCORE Search Results Details for Application 10553669 and Search Result 20071121_092710_us-10-553-669-4.rup.

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                        GenCore version 6.2.1
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OM protein - protein search, using sw model

Run on:         November 21, 2007, 09:27:44 ; Search time 150 Seconds
                                        (without alignments)
                                        2284.144 Million cell updates/sec

Title:          US-10-553-669-4
Perfect score:  1711
Sequence:       1 PCPGACVCYNEPKVTTSCPQ.........TDEEPLGLPKCCQPDAADKA 319

Scoring table:  BLOSUM62
                Gapop 10.0 , Gapext 0.5

Searched:       3281787 seqs, 1072124677 residues

Total number of hits satisfying chosen parameters:      3281787

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
                 Maximum Match 100%
                 Listing first 45 summaries

Database :       UniProt_8.4:*
                1:  uniprot_sprot:*
                2:  uniprot_trembl:*

     Pred. No. is the number of results predicted by chance to have a
     score greater than or equal to the score of the result being printed,
     and is derived by analysis of the total score distribution.

                                SUMMARIES
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Result No.	Score	% Query Match	Length	DB	ID	Description
1	1711	100.0	473	1	RTN4R_HUMAN	Q9bzr6 homo sapien
2	1670	97.6	473	1	RTN4R_MACFA	Q9n0e3 macaca fasc
3	1531	89.5	473	1	RTN4R_MOUSE	Q99pi8 mus musculu
4	1529	89.4	473	1	RTN4R_RAT	Q99m75 rattus norv
5	927.5	54.2	479	2	Q6DH76_BRARE	Q6dh76 brachydanio
6	927.5	54.2	479	2	Q6X3Y5_BRARE	Q6x3y5 brachydanio
7	774.5	45.3	412	2	Q4RRU8_TETNG	Q4rru8 tetraodon n
8	774	45.2	420	1	R4RL2_MOUSE	Q7m6z0 mus musculu

9	773.5	45.2	478	2	Q6WZD2_BRARE	Q6wzd2 brachydanio
10	773	45.2	420	1	R4RL2_HUMAN	Q86un3 homo sapien
11	773	45.2	420	2	Q17RL9_HUMAN	Q17rl9 homo sapien
12	772	45.1	441	1	R4RL1_HUMAN	Q86un2 homo sapien
13	771	45.1	420	1	R4RL2_RAT	Q80wd1 rattus norv
14	760	44.4	445	1	R4RL1_MOUSE	Q8k0s5 mus musculu
15	756	44.2	445	1	R4RL1_RAT	Q80wd0 rattus norv
16	755.5	44.2	457	2	Q6WZD1_BRARE	Q6wzd1 brachydanio
17	749.5	43.8	310	2	Q4RRQ4_TETNG	Q4rrq4 tetraodon n
18	705	41.2	324	2	Q4S3K9_TETNG	Q4s3k9 tetraodon n
19	698	40.8	411	2	Q4S6L6_TETNG	Q4s6l6 tetraodon n
20	672.5	39.3	458	2	Q6WZD3_BRARE	Q6wzd3 brachydanio
21	374	21.9	411	2	Q4S9P3_TETNG	Q4s9p3 tetraodon n
22	373	21.8	466	2	Q66IW3_XENLA	Q66iw3 xenopus lae
23	372.5	21.8	762	2	Q5JY13_HUMAN	Q5jy13 homo sapien
24	372.5	21.8	778	2	Q6NUI6_HUMAN	Q6nui6 homo sapien
25	367.5	21.5	453	2	Q86XY1_HUMAN	Q86xy1 homo sapien
26	365.5	21.4	692	2	Q4G0S0_HUMAN	Q4g0s0 homo sapien
27	364.5	21.3	481	1	NYX_HUMAN	Q9gzu5 homo sapien
28	364.5	21.3	481	2	Q2M1S4_HUMAN	Q2m1s4 homo sapien
29	357.5	20.9	935	2	Q4SBT7_TETNG	Q4sbt7 tetraodon n
30	354	20.7	417	2	Q6E4J7_PETMA	Q6e4j7 petromyzon
31	352	20.6	339	2	Q4SU68_TETNG	Q4su68 tetraodon n
32	351.5	20.5	652	2	Q45R42_RAT	Q45r42 rattus norv
33	351	20.5	606	2	Q1KS52_PIG	Q1ks52 sus scrofa
34	350.5	20.5	513	1	LRC24_HUMAN	Q50lg9 homo sapien
35	349.5	20.4	476	1	NYX_MOUSE	P83503 mus musculu
36	349.5	20.4	652	1	LRRC4_MOUSE	Q99ph1 mus musculu
37	349.5	20.4	653	1	LRRC4_HUMAN	Q9hbw1 homo sapien
38	346.5	20.3	597	2	Q3I0Y3_BOVIN	Q3i0y3 bos taurus
39	346.5	20.3	602	2	Q58CS0_BOVIN	Q58cs0 bos taurus
40	345.5	20.2	640	2	Q4JIW0_HUMAN	Q4jiw0 homo sapien
41	344.5	20.1	521	1	LRC24_MOUSE	Q8bha1 mus musculu
42	343.5	20.1	605	2	Q8TAY0_HUMAN	Q8tay0 homo sapien
43	343.5	20.1	640	1	NGL1_HUMAN	Q9hcj2 homo sapien
44	343.5	20.1	640	1	NGL1_MOUSE	Q8c031 mus musculu
45	343.5	20.1	640	2	Q505E5_MOUSE	Q505e5 mus musculu

ALIGNMENTS

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RESULT 1
RTN4R_HUMAN
ID   RTN4R_HUMAN     STANDARD;      PRT;   473 AA.
AC   Q9BZR6;
DT   25-NOV-2002, integrated into UniProtKB/Swiss-Prot.
DT   01-JUN-2001, sequence version 1.
DT   27-JUN-2006, entry version 54.
DE   Reticulon-4 receptor precursor (Nogo receptor) (NgR) (Nogo-66
DE   receptor).
GN   Name=RTN4R; Synonyms=NOGOR; ORFNames=UNQ330/PRO526;
OS   Homo sapiens (Human).
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC   Mammalia; Eutheria; Euarchontoglires; Primates; Haplorrhini;
OC   Catarrhini; Hominidae; Homo.
OX   NCBI_TaxID=9606;
RN   [1]
RP   NUCLEOTIDE SEQUENCE [MRNA].
RC   TISSUE=Brain;
RX   MEDLINE=21069055; PubMed=11201742; DOI=10.1038/35053072;
RA   Fournier A.E., GrandPre T., Strittmatter S.M.;
RT   "Identification of a receptor mediating Nogo-66 inhibition of axonal
RT   regeneration.";
RL   Nature 409:341-346(2001).
```

SCORE Search Results Details for Application 10553669 and Search Result 20071121_092710_us-10-553-669-5.rup.

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GenCore version 6.2.1

OM protein - protein search, using sw model

Run on: November 21, 2007, 09:27:44 ; Search time 133 Seconds
(without alignments)
2284.144 Million cell updates/sec

Title: US-10-553-669-5
Perfect score: 1511
Sequence: 1 CPGACVCYNEPKVTTSRPQQ.....QRLAGRDLKRLATSDLEGCA 284

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 3281787 seqs, 1072124677 residues

Total number of hits satisfying chosen parameters: 3281787

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database : UniProt_8.4:*
1: uniprot_sprot:*
2: uniprot_trembl:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	1481	98.0	473	1	RTN4R_RAT	Q99m75 rattus norv
2	1444	95.6	473	1	RTN4R_MOUSE	Q99pi8 mus musculu
3	1352	89.5	473	1	RTN4R_MACFA	Q9n0e3 macaca fasc
4	1351	89.4	473	1	RTN4R_HUMAN	Q9bzr6 homo sapien
5	910	60.2	479	2	Q6DH76_BRARE	Q6dh76 brachydanio
6	910	60.2	479	2	Q6X3Y5_BRARE	Q6x3y5 brachydanio
7	730.5	48.3	478	2	Q6WZD2_BRARE	Q6wzd2 brachydanio
8	729.5	48.3	420	1	R4RL2_HUMAN	Q86un3 homo sapien

9	729.5	48.3	420	2	Q17RL9_HUMAN	Q17rl9 homo sapien
10	726.5	48.1	457	2	Q6WZD1_BRARE	Q6wzd1 brachydanio
11	725.5	48.0	441	1	R4RL1_HUMAN	Q86un2 homo sapien
12	721.5	47.7	412	2	Q4RRU8_TETNG	Q4rru8 tetraodon n
13	721.5	47.7	420	1	R4RL2_MOUSE	Q7m6z0 mus musculu
14	717.5	47.5	445	1	R4RL1_MOUSE	Q8k0s5 mus musculu
15	716.5	47.4	420	1	R4RL2_RAT	Q80wd1 rattus norv
16	714.5	47.3	310	2	Q4RRQ4_TETNG	Q4rrq4 tetraodon n
17	712.5	47.2	445	1	R4RL1_RAT	Q80wd0 rattus norv
18	677	44.8	324	2	Q4S3K9_TETNG	Q4s3k9 tetraodon n
19	657.5	43.5	411	2	Q4S6L6_TETNG	Q4s6l6 tetraodon n
20	631.5	41.8	458	2	Q6WZD3_BRARE	Q6wzd3 brachydanio
21	360.5	23.9	652	2	Q45R42_RAT	Q45r42 rattus norv
22	357.5	23.7	652	1	LRRC4_MOUSE	Q99ph1 mus musculu
23	357.5	23.7	653	1	LRRC4_HUMAN	Q9hbw1 homo sapien
24	357.5	23.7	762	2	Q5JY13_HUMAN	Q5jy13 homo sapien
25	357.5	23.7	778	2	Q6NUI6_HUMAN	Q6nui6 homo sapien
26	354.5	23.5	597	2	Q3I0Y3_BOVIN	Q3i0y3 bos taurus
27	354.5	23.5	602	2	Q58CS0_BOVIN	Q58cs0 bos taurus
28	352	23.3	411	2	Q4S9P3_TETNG	Q4s9p3 tetraodon n
29	350.5	23.2	692	2	Q4G0S0_HUMAN	Q4g0s0 homo sapien
30	350	23.2	466	2	Q66IW3_XENLA	Q66iw3 xenopus lae
31	350	23.2	935	2	Q4SBT7_TETNG	Q4sbt7 tetraodon n
32	342.5	22.7	709	1	LRC4B_MOUSE	P0c192 mus musculu
33	338.5	22.4	481	1	NYX_HUMAN	Q9gzu5 homo sapien
34	338.5	22.4	481	2	Q2M1S4_HUMAN	Q2m1s4 homo sapien
35	338.5	22.4	713	1	LRC4B_HUMAN	Q9nt99 homo sapien
36	337.5	22.3	640	2	Q4JIW0_HUMAN	Q4jiw0 homo sapien
37	335.5	22.2	640	1	NGL1_HUMAN	Q9hcj2 homo sapien
38	335.5	22.2	640	1	NGL1_MOUSE	Q8c031 mus musculu
39	335.5	22.2	640	2	Q505E5_MOUSE	Q505e5 mus musculu
40	334	22.1	417	2	Q6E4J7_PETMA	Q6e4j7 petromyzon
41	333.5	22.1	339	2	Q4SU68_TETNG	Q4su68 tetraodon n
42	330.5	21.9	370	2	Q2YE77_EPTST	Q2ye77 eptatretus
43	330.5	21.9	370	2	Q2YE78_EPTST	Q2ye78 eptatretus
44	328.5	21.7	393	2	Q32R29_EPTBU	Q32r29 eptatretus
45	327.5	21.7	257	2	Q2VGP9_PETMA	Q2vgp9 petromyzon

ALIGNMENTS

```
RESULT 1
RTN4R_RAT
ID   RTN4R_RAT      STANDARD;      PRT;   473 AA.
AC   Q99M75;
DT   25-NOV-2002, integrated into UniProtKB/Swiss-Prot.
DT   10-MAY-2005, sequence version 2.
DT   27-JUN-2006, entry version 41.
DE   Reticulon-4 receptor precursor (Nogo receptor) (NgR) (Nogo-66
DE   receptor).
GN   Name=Rtn4r; Synonyms=Nogor;
OS   Rattus norvegicus (Rat).
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC   Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Sciurognathi;
OC   Muroidea; Muridae; Murinae; Rattus.
OX   NCBI_TaxID=10116;
RN   [1]
RP   NUCLEOTIDE SEQUENCE [MRNA].
RC   STRAIN=Sprague-Dawley;
RA   Jin W.-L., Jia W., Long M., Ju G.;
RT   "Identification and preparation of polyclonal antibody against rat
RT   Nogo receptor.";
RL   Submitted (MAR-2001) to the EMBL/GenBank/DDBJ databases.
RN   [2]
```

SCORE Search Results Details for Application 10553669 and Search Result 20071121_092710_us-10-553-669-6.rup.

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This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092710_us-10-553-669-6.rup.

Go Back to previous page

```
                        GenCore version 6.2.1
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OM protein - protein search, using sw model

Run on:          November 21, 2007, 09:27:44 ; Search time 149 Seconds
                                            (without alignments)
                                            2284.144 Million cell updates/sec

Title:           US-10-553-669-6
Perfect score:   1695
Sequence:        1 CPGACVCYNEPKVTTSRPQQ.........TDEELLGLPKCCQPDAADKA 318

Scoring table:   BLOSUM62
                 Gapop 10.0 , Gapext 0.5

Searched:        3281787 seqs, 1072124677 residues

Total number of hits satisfying chosen parameters:      3281787

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
                 Maximum Match 100%
                 Listing first 45 summaries

Database :        UniProt_8.4:*
                 1:  uniprot_sprot:*
                 2:  uniprot_trembl:*

      Pred. No. is the number of results predicted by chance to have a
      score greater than or equal to the score of the result being printed,
      and is derived by analysis of the total score distribution.

                                SUMMARIES
```

Result No.	Score	% Query Match	Length	DB	ID	Description
1	1665	98.2	473	1	RTN4R_RAT	Q99m75 rattus norv
2	1611	95.0	473	1	RTN4R_MOUSE	Q99pi8 mus musculu
3	1492	88.0	473	1	RTN4R_MACFA	Q9n0e3 macaca fasc
4	1489	87.8	473	1	RTN4R_HUMAN	Q9bzr6 homo sapien
5	930.5	54.9	479	2	Q6DH76_BRARE	Q6dh76 brachydanio
6	930.5	54.9	479	2	Q6X3Y5_BRARE	Q6x3y5 brachydanio
7	738	43.5	420	1	R4RL2_HUMAN	Q86un3 homo sapien
8	738	43.5	420	2	Q17RL9_HUMAN	Q17rl9 homo sapien

9	737	43.5	441	1	R4RL1_HUMAN	Q86un2 homo sapien
10	731.5	43.2	478	2	Q6WZD2_BRARE	Q6wzd2 brachydanio
11	730	43.1	445	1	R4RL1_MOUSE	Q8k0s5 mus musculu
12	729.5	43.0	412	2	Q4RRU8_TETNG	Q4rru8 tetraodon n
13	726.5	42.9	457	2	Q6WZD1_BRARE	Q6wzd1 brachydanio
14	725	42.8	420	1	R4RL2_MOUSE	Q7m6z0 mus musculu
15	724	42.7	445	1	R4RL1_RAT	Q80wd0 rattus norv
16	720	42.5	420	1	R4RL2_RAT	Q80wd1 rattus norv
17	714.5	42.2	310	2	Q4RRQ4_TETNG	Q4rrq4 tetraodon n
18	677	39.9	324	2	Q4S3K9_TETNG	Q4s3k9 tetraodon n
19	664	39.2	411	2	Q4S6L6_TETNG	Q4s616 tetraodon n
20	631.5	37.3	458	2	Q6WZD3_BRARE	Q6wzd3 brachydanio
21	367	21.7	652	2	Q45R42_RAT	Q45r42 rattus norv
22	362	21.4	652	1	LRRC4_MOUSE	Q99ph1 mus musculu
23	361.5	21.3	653	1	LRRC4_HUMAN	Q9hbw1 homo sapien
24	358.5	21.2	597	2	Q3I0Y3_BOVIN	Q3i0y3 bos taurus
25	358.5	21.2	602	2	Q58CS0_BOVIN	Q58cs0 bos taurus
26	358.5	21.2	762	2	Q5JY13_HUMAN	Q5jy13 homo sapien
27	358.5	21.2	778	2	Q6NUI6_HUMAN	Q6nui6 homo sapien
28	355.5	21.0	935	2	Q4SBT7_TETNG	Q4sbt7 tetraodon n
29	354	20.9	411	2	Q4S9P3_TETNG	Q4s9p3 tetraodon n
30	351.5	20.7	692	2	Q4G0S0_HUMAN	Q4g0s0 homo sapien
31	350	20.6	466	2	Q66IW3_XENLA	Q66iw3 xenopus lae
32	342.5	20.2	709	1	LRC4B_MOUSE	P0c192 mus musculu
33	338.5	20.0	481	1	NYX_HUMAN	Q9gzu5 homo sapien
34	338.5	20.0	481	2	Q2M1S4_HUMAN	Q2m1s4 homo sapien
35	338.5	20.0	713	1	LRC4B_HUMAN	Q9nt99 homo sapien
36	337.5	19.9	640	2	Q4JIW0_HUMAN	Q4jiw0 homo sapien
37	335.5	19.8	640	1	NGL1_HUMAN	Q9hcj2 homo sapien
38	335.5	19.8	640	1	NGL1_MOUSE	Q8c031 mus musculu
39	335.5	19.8	640	2	Q505E5_MOUSE	Q505e5 mus musculu
40	335	19.8	1529	2	Q7ZXI2_XENLA	Q7zxi2 xenopus lae
41	334	19.7	417	2	Q6E4J7_PETMA	Q6e4j7 petromyzon
42	333.5	19.7	339	2	Q4SU68_TETNG	Q4su68 tetraodon n
43	333	19.6	782	2	Q5T0V4_HUMAN	Q5t0v4 homo sapien
44	333	19.6	1461	2	Q5VW18_HUMAN	Q5vw18 homo sapien
45	333	19.6	1534	1	SLIT1_HUMAN	O75093 homo sapien

ALIGNMENTS

```
RESULT 1
RTN4R_RAT
ID   RTN4R_RAT      STANDARD;      PRT;   473 AA.
AC   Q99M75;
DT   25-NOV-2002, integrated into UniProtKB/Swiss-Prot.
DT   10-MAY-2005, sequence version 2.
DT   27-JUN-2006, entry version 41.
DE   Reticulon-4 receptor precursor (Nogo receptor) (NgR) (Nogo-66
DE   receptor).
GN   Name=Rtn4r; Synonyms=Nogor;
OS   Rattus norvegicus (Rat).
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC   Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Sciurognathi;
OC   Muroidea; Muridae; Murinae; Rattus.
OX   NCBI_TaxID=10116;
RN   [1]
RP   NUCLEOTIDE SEQUENCE [MRNA].
RC   STRAIN=Sprague-Dawley;
RA   Jin W.-L., Jia W., Long M., Ju G.;
RT   "Identification and preparation of polyclonal antibody against rat
RT   Nogo receptor.";
RL   Submitted (MAR-2001) to the EMBL/GenBank/DDBJ databases.
RN   [2]
```

SCORE Search Results D

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This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

```
                          GenCore version 6.2.1
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OM protein - protein search, using sw model

Run on:         November 21, 2007, 09:43:31 ; Search time 17 Seconds
                                        (without alignments)
                                        1938.665 Million cell updates/sec

Title:          US-10-553-669-1
Perfect score:  1842
Sequence:       1 MKRASAGGSRLLAWVLWLQA.........TDEEPLGLPKCCQPDAADKA 344

Scoring table:  BLOSUM62
                Gapop 10.0 , Gapext 0.5

Searched:       283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters:      283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
                 Maximum Match 100%
                 Listing first 45 summaries

Database :       PIR_80:*
                1:  pir1:*
                2:  pir2:*
                3:  pir3:*
                4:  pir4:*

     Pred. No. is the number of results predicted by chance to have a
     score greater than or equal to the score of the result being printed,
     and is derived by analysis of the total score distribution.

                                SUMMARIES
```

Result No.	Score	% Query Match	Length	DB	ID	Description
1	367	19.9	1531	2	T42218	slit-1 protein hom
2	342.5	18.6	605	2	A41915	insulin-like growt
3	336	18.2	1523	2	T13953	MEGF5 protein - ra
4	330.5	17.9	605	2	JC5239	insulin-like growt
5	327	17.8	1469	2	B36665	slit protein 2 pre
6	327	17.8	1480	2	A36665	slit protein 1 pre
7	321.5	17.5	622	2	JC7973	synleurin - human
8	310	16.8	603	2	JC6128	insulin-like growt
9	309.5	16.8	361	2	A53860	chondroadherin pre
10	304	16.5	560	2	A60164	platelet membrane
11	299.5	16.3	603	2	JC1282	insulin-like growt

12	295	16.0	907	2	JG0193	G protein-coupled
13	291	15.8	1091	2	A58532	glial cell membran
14	290.5	15.8	907	2	JE0176	orphan G protein-c
15	287.5	15.6	536	2	A34901	lysine carboxypept
16	278.5	15.1	420	2	A53531	oncofetal trophobl
17	261	14.2	312	1	NBHUA2	leucine-rich alpha
18	256.5	13.9	707	2	JC7763	neuronal leucine-r
19	246.5	13.4	359	1	NBHUC8	decorin precursor
20	241	13.1	1025	2	T42626	secreted leucine-r
21	238.5	12.9	357	2	S24317	decorin precursor
22	234.5	12.7	682	2	A49121	cell-surface molec
23	234.5	12.7	682	2	A43318	connectin precurso
24	233.5	12.7	360	2	S06280	decorin precursor
25	230.5	12.5	360	2	I47020	decorin - rabbit
26	230	12.5	789	2	T28714	hypothetical prote
27	230	12.5	1355	2	T28715	hypothetical prote
28	227.5	12.4	1535	2	S46224	peroxidasin - frui
29	227	12.3	333	2	T34555	hypothetical prote
30	226.5	12.3	594	2	T23841	hypothetical prote
31	226	12.3	1389	2	T13852	gene wheeler prote
32	224	12.2	1385	2	T13887	tlr protein - frui
33	221.5	12.0	354	2	A55454	decorin precursor
34	218.5	11.9	610	2	T23836	hypothetical prote
35	215.5	11.7	354	2	S29145	decorin precursor
36	208	11.3	382	2	I39068	proline- arginine-
37	208	11.3	1112	2	T10504	disease resistance
38	206.5	11.2	562	2	T34319	hypothetical prote
39	205.5	11.2	1066	2	T15864	hypothetical prote
40	204	11.1	738	2	T19938	hypothetical prote
41	202	11.0	342	2	A46743	lumican precursor
42	202	11.0	662	2	S42799	garp precursor - h
43	200	10.9	338	2	S52284	lumicon, secretory
44	200	10.9	369	2	S32559	biglycan precursor
45	199.5	10.8	626	1	NBHUIA	platelet glycoprot

ALIGNMENTS

```
RESULT 1
T42218
slit-1 protein homolog - rat
N;Alternate names: MEGF4 protein
C;Species: Rattus norvegicus (Norway rat)
C;Date: 03-Dec-1999 #sequence_revision 03-Dec-1999 #text_change 09-Jul-2004
C;Accession: T42218
R;Nakayama, M.; Nakajima, D.; Nagase, T.; Nomura, N.; Seki, N.; Ohara, O.
Genomics 51, 27-34, 1998
A;Title: Identification of high-molecular-weight proteins with multiple EGF-like motifs by motif-tra
A;Reference number: Z14126; MUID:98360089; PMID:9693030
A;Accession: T42218
A;Status: preliminary; translated from GB/EMBL/DDBJ
A;Molecule type: mRNA
A;Residues: 1-1531 <NAK>
A;Cross-references: UNIPROT:O88279; UNIPARC:UPI000004F20B; EMBL:AB011530; NID:g3449289; PIDN:BAA3246
A;Experimental source: strain Sprague-Dawley; brain
C;Genetics:
A;Gene: MEGF4
C;Superfamily: fruit fly slit protein; EGF homology; leucine-rich alpha-2-glycoprotein repeat homolo

  Query Match             19.9%;  Score 367;  DB 2;  Length 1531;
  Best Local Similarity   21.9%;  Pred. No. 2.3e-23;
  Matches  110;  Conservative   62;  Mismatches  133;  Indels  198;  Gaps   10;

Qy          4 ASAGGSRLLAW-VLWLQAWQV-AAPCPGACVCYNEPKVTTSCPQQGLQAVPVGIPAASQR 61
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SCORE Search Results D

This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

GenCore version 6.2.1
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DM protein - protein search, using sw model

Run on: November 21, 2007, 09:43:31 ; Search time 17 Seconds
(without alignments)
1938.665 Million cell updates/sec

Title: US-10-553-669-2
Perfect score: 1838
Sequence: 1 MKRASSGGSRLPTWVLWLQA.....TDEELLGLPKCCQPDAADKA 344

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters: 283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database : PIR_80:*
1: pir1:*
2: pir2:*
3: pir3:*
4: pir4:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	369	20.1	1531	2	T42218	slit-1 protein hom
2	328	17.8	622	2	JC7973	synleurin - human
3	319.5	17.4	605	2	A41915	insulin-like growt
4	318	17.3	1469	2	B36665	slit protein 2 pre
5	318	17.3	1480	2	A36665	slit protein 1 pre
6	318	17.3	1523	2	T13953	MEGF5 protein - ra
7	313.5	17.1	605	2	JC5239	insulin-like growt
8	293.5	16.0	907	2	JE0176	orphan G protein-c
9	293	15.9	603	2	JC6128	insulin-like growt
10	292	15.9	603	2	JC1282	insulin-like growt
11	283	15.4	560	2	A60164	platelet membrane

12	279.5	15.2	361	2	A53860	chondroadherin pre
13	278	15.1	907	2	JG0193	G protein-coupled
14	277	15.1	1091	2	A58532	glial cell membran
15	266	14.5	536	2	A34901	lysine carboxypept
16	250	13.6	420	2	A53531	oncofetal trophobl
17	243	13.2	312	1	NBHUA2	leucine-rich alpha
18	235.5	12.8	594	2	T23841	hypothetical prote
19	234	12.7	707	2	JC7763	neuronal leucine-r
20	230.5	12.5	359	1	NBHUC8	decorin precursor
21	229	12.5	682	2	A49121	cell-surface molec
22	229	12.5	682	2	A43318	connectin precurso
23	227.5	12.4	360	2	S06280	decorin precursor
24	227.5	12.4	610	2	T23836	hypothetical prote
25	221.5	12.1	360	2	I47020	decorin - rabbit
26	220	12.0	1389	2	T13852	gene wheeler prote
27	218	11.9	1385	2	T13887	tlr protein - frui
28	217.5	11.8	357	2	S24317	decorin precursor
29	214.5	11.7	354	2	A55454	decorin precursor
30	213.5	11.6	653	2	T25194	hypothetical prote
31	213	11.6	333	2	T34555	hypothetical prote
32	213	11.6	789	2	T28714	hypothetical prote
33	213	11.6	1355	2	T28715	hypothetical prote
34	209	11.4	382	2	I39068	proline- arginine-
35	207	11.3	562	2	T34319	hypothetical prote
36	204.5	11.1	738	2	T19938	hypothetical prote
37	203.5	11.1	354	2	S29145	decorin precursor
38	203.5	11.1	1016	2	T30553	disease resistance
39	203.5	11.1	1535	2	S46224	peroxidasin - frui
40	203	11.0	1025	2	T42626	secreted leucine-r
41	202.5	11.0	1112	2	T10504	disease resistance
42	200.5	10.9	1066	2	T15864	hypothetical prote
43	196.5	10.7	375	2	S05390	fibromodulin precu
44	196	10.7	961	2	T23395	hypothetical prote
45	195.5	10.6	680	2	T19939	hypothetical prote

ALIGNMENTS

RESULT 1
T42218
slit-1 protein homolog - rat
N;Alternate names: MEGF4 protein
C;Species: Rattus norvegicus (Norway rat)
C;Date: 03-Dec-1999 #sequence_revision 03-Dec-1999 #text_change 09-Jul-2004
C;Accession: T42218
R;Nakayama, M.; Nakajima, D.; Nagase, T.; Nomura, N.; Seki, N.; Ohara, O.
Genomics 51, 27-34, 1998
A;Title: Identification of high-molecular-weight proteins with multiple EGF-like motifs by motif-tra
A;Reference number: Z14126; MUID:98360089; PMID:9693030
A;Accession: T42218
A;Status: preliminary; translated from GB/EMBL/DDBJ
A;Molecule type: mRNA
A;Residues: 1-1531 <NAK>
A;Cross-references: UNIPROT:O88279; UNIPARC:UPI000004F20B; EMBL:AB011530; NID:g3449289; PIDN:BAA324
A;Experimental source: strain Sprague-Dawley; brain
C;Genetics:
A;Gene: MEGF4
C;Superfamily: fruit fly slit protein; EGF homology; leucine-rich alpha-2-glycoprotein repeat homolo

Query Match 20.1%; Score 369; DB 2; Length 1531;
Best Local Similarity 22.4%; Pred. No. 9.5e-24;
Matches 117; Conservative 62; Mismatches 150; Indels 194; Gaps 11;

Qy 4 ASSGGSRLPTW-VLWLQAWRV-ATPCPGACVCYNEPKVTTSRPQQGLQAVPAGIPASSQR 61

SCORE Search Results D

This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

GenCore version 6.2.1
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OM protein - protein search, using sw model

Run on: November 21, 2007, 09:43:31 ; Search time 14 Seconds
(without alignments)
1938.665 Million cell updates/sec

Title: US-10-553-669-3
Perfect score: 1515
Sequence: 1 PCPGACVCYNEPKVTTSCPQ.....QRLAGRDLKRLAANDLQGCA 285

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters: 283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database : PIR_80:*
1: pir1:*
2: pir2:*
3: pir3:*
4: pir4:*

Pred. No. is the number of results predicted by chance to have a
score greater than or equal to the score of the result being printed,
and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	342.5	22.6	605	2	A41915	insulin-like growt
2	330	21.8	1531	2	T42218	slit-1 protein hom
3	329	21.7	1523	2	T13953	MEGF5 protein - ra
4	328.5	21.7	605	2	JC5239	insulin-like growt
5	325	21.5	1469	2	B36665	slit protein 2 pre
6	325	21.5	1480	2	A36665	slit protein 1 pre
7	318.5	21.0	622	2	JC7973	synleurin - human
8	305	20.1	603	2	JC6128	insulin-like growt
9	304.5	20.1	361	2	A53860	chondroadherin pre
10	297	19.6	603	2	JC1282	insulin-like growt
11	295	19.5	907	2	JG0193	G protein-coupled

12	290	19.1	907	2	JE0176	orphan G protein-c
13	289.5	19.1	560	2	A60164	platelet membrane
14	287	18.9	536	2	A34901	lysine carboxypept
15	276.5	18.3	420	2	A53531	oncofetal trophobl
16	276	18.2	1091	2	A58532	glial cell membran
17	261	17.2	312	1	NBHUA2	leucine-rich alpha
18	250.5	16.5	707	2	JC7763	neuronal leucine-r
19	246.5	16.3	359	1	NBHUC8	decorin precursor
20	241	15.9	1025	2	T42626	secreted leucine-r
21	238.5	15.7	357	2	S24317	decorin precursor
22	234.5	15.5	682	2	A49121	cell-surface molec
23	234.5	15.5	682	2	A43318	connectin precurso
24	233.5	15.4	360	2	S06280	decorin precursor
25	230.5	15.2	360	2	I47020	decorin - rabbit
26	230	15.2	789	2	T28714	hypothetical prote
27	230	15.2	1355	2	T28715	hypothetical prote
28	227	15.0	333	2	T34555	hypothetical prote
29	226	14.9	594	2	T23841	hypothetical prote
30	226	14.9	1389	2	T13852	gene wheeler prote
31	223	14.7	1385	2	T13887	tlr protein - frui
32	221.5	14.6	354	2	A55454	decorin precursor
33	219.5	14.5	1535	2	S46224	peroxidasin - frui
34	218	14.4	610	2	T23836	hypothetical prote
35	215.5	14.2	354	2	S29145	decorin precursor
36	208	13.7	382	2	I39068	proline- arginine-
37	206	13.6	1112	2	T10504	disease resistance
38	205.5	13.6	1066	2	T15864	hypothetical prote
39	204	13.5	738	2	T19938	hypothetical prote
40	202	13.3	342	2	A46743	lumican precursor
41	202	13.3	562	2	T34319	hypothetical prote
42	202	13.3	662	2	S42799	garp precursor - h
43	199.5	13.2	626	1	NBHUIA	platelet glycoprot
44	199	13.1	338	2	S52284	lumicon, secretory
45	197.5	13.0	961	2	T23395	hypothetical prote

ALIGNMENTS

RESULT 1
A41915
insulin-like growth factor-binding complex acid-labile chain precursor - human
N;Alternate names: Acid-Labile Subunit (ALS)
C;Species: Homo sapiens (man)
C;Date: 31-Dec-1993 #sequence_revision 31-Dec-1993 #text_change 09-Jul-2004
C;Accession: A41915
R;Leong, S.R.; Baxter, R.C.; Camerato, T.; Dai, J.; Wood, W.I.
Mol. Endocrinol. 6, 870-876, 1992
A;Title: Structure and functional expression of the acid-labile subunit of the insulin-like growth f
A;Reference number: A41915; MUID:92357025; PMID:1379671
A;Accession: A41915
A;Status: preliminary
A;Molecule type: mRNA; protein
A;Residues: 1-605 <LEO>
A;Cross-references: UNIPROT:P35858; UNIPARC:UPI000000088A; GB:M86826; NID:g184807; PIDN:AAA36047.1;
A;Experimental source: liver
A;Note: sequence extracted from NCBI backbone (NCBIP:110171)
F;75-98/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR1>
F;99-122/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR2>
F;123-146/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR3>
F;147-170/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR4>
F;171-194/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR5>
F;195-218/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR6>
F;219-242/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR7>
F;243-266/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR8>

SCORE Search Results D

This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

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OM protein - protein search, using sw model

Run on: November 21, 2007, 09:43:31 ; Search time 16 Seconds
(without alignments)
1938.665 Million cell updates/sec

Title: US-10-553-669-4
Perfect score: 1711
Sequence: 1 PCPGACVCYNEPKVTTSCPQ.....TDEEPLGLPKCCQPDAADKA 319

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters: 283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database : PIR_80:*
1: pir1:*
2: pir2:*
3: pir3:*
4: pir4:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	342.5	20.0	605	2	A41915	insulin-like growt
2	333	19.5	1531	2	T42218	slit-1 protein hom
3	329	19.2	1523	2	T13953	MEGF5 protein - ra
4	328.5	19.2	605	2	JC5239	insulin-like growt
5	325	19.0	1469	2	B36665	slit protein 2 pre
6	325	19.0	1480	2	A36665	slit protein 1 pre
7	321.5	18.8	622	2	JC7973	synleurin - human
8	306.5	17.9	361	2	A53860	chondroadherin pre
9	305	17.8	603	2	JC6128	insulin-like growt
10	304	17.8	560	2	A60164	platelet membrane
11	297	17.4	603	2	JC1282	insulin-like growt

12	295	17.2	907	2	JG0193	G protein-coupled
13	290	16.9	907	2	JE0176	orphan G protein-c
14	287.5	16.8	536	2	A34901	lysine carboxypept
15	276.5	16.2	420	2	A53531	oncofetal trophobl
16	276	16.1	1091	2	A58532	glial cell membran
17	261	15.3	312	1	NBHUA2	leucine-rich alpha
18	250.5	14.6	707	2	JC7763	neuronal leucine-r
19	246.5	14.4	359	1	NBHUC8	decorin precursor
20	241	14.1	1025	2	T42626	secreted leucine-r
21	238.5	13.9	357	2	S24317	decorin precursor
22	234.5	13.7	682	2	A49121	cell-surface molec
23	234.5	13.7	682	2	A43318	connectin precurso
24	233.5	13.6	360	2	S06280	decorin precursor
25	230.5	13.5	360	2	I47020	decorin - rabbit
26	230	13.4	789	2	T28714	hypothetical prote
27	230	13.4	1355	2	T28715	hypothetical prote
28	227	13.3	333	2	T34555	hypothetical prote
29	226.5	13.2	594	2	T23841	hypothetical prote
30	226	13.2	1389	2	T13852	gene wheeler prote
31	223	13.0	1385	2	T13887	tlr protein - frui
32	222.5	13.0	1535	2	S46224	peroxidasin - frui
33	221.5	12.9	354	2	A55454	decorin precursor
34	218.5	12.8	610	2	T23836	hypothetical prote
35	215.5	12.6	354	2	S29145	decorin precursor
36	208	12.2	382	2	I39068	proline- arginine-
37	206.5	12.1	562	2	T34319	hypothetical prote
38	206	12.0	1112	2	T10504	disease resistance
39	205.5	12.0	1066	2	T15864	hypothetical prote
40	204	11.9	738	2	T19938	hypothetical prote
41	202	11.8	342	2	A46743	lumican precursor
42	202	11.8	662	2	S42799	garp precursor - h
43	199.5	11.7	626	1	NBHUIA	platelet glycoprot
44	199	11.6	338	2	S52284	lumicon, secretory
45	197.5	11.5	961	2	T23395	hypothetical prote

ALIGNMENTS

RESULT 1
A41915
insulin-like growth factor-binding complex acid-labile chain precursor - human
N;Alternate names: Acid-Labile Subunit (ALS)
C;Species: Homo sapiens (man)
C;Date: 31-Dec-1993 #sequence_revision 31-Dec-1993 #text_change 09-Jul-2004
C;Accession: A41915
R;Leong, S.R.; Baxter, R.C.; Camerato, T.; Dai, J.; Wood, W.I.
Mol. Endocrinol. 6, 870-876, 1992
A;Title: Structure and functional expression of the acid-labile subunit of the insulin-like growth f
A;Reference number: A41915; MUID:92357025; PMID:1379671
A;Accession: A41915
A;Status: preliminary
A;Molecule type: mRNA; protein
A;Residues: 1-605 <LEO>
A;Cross-references: UNIPROT:P35858; UNIPARC:UPI000000088A; GB:M86826; NID:g184807; PIDN:AAA36047.1;
A;Experimental source: liver
A;Note: sequence extracted from NCBI backbone (NCBIP:110171)
F;75-98/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR1>
F;99-122/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR2>
F;123-146/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR3>
F;147-170/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR4>
F;171-194/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR5>
F;195-218/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR6>
F;219-242/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR7>
F;243-266/Domain: leucine-rich alpha-2-glycoprotein repeat homology <LRR8>

SCORE Search Results D

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This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

```
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OM protein - protein search, using sw model

Run on:         November 21, 2007, 09:43:31 ; Search time 14 Seconds
                                        (without alignments)
                                        1938.665 Million cell updates/sec

Title:          US-10-553-669-5
Perfect score:  1511
Sequence:       1 CPGACVCYNEPKVTTSRPQQ.........QRLAGRDLKRLATSDLEGCA 284

Scoring table:  BLOSUM62
                Gapop 10.0 , Gapext 0.5

Searched:       283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters:      283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
                 Maximum Match 100%
                 Listing first 45 summaries

Database :       PIR_80:*
                1:  pir1:*
                2:  pir2:*
                3:  pir3:*
                4:  pir4:*

     Pred. No. is the number of results predicted by chance to have a
     score greater than or equal to the score of the result being printed,
     and is derived by analysis of the total score distribution.

                                SUMMARIES
```

Result No.	Score	% Query Match	Length	DB	ID	Description
1	323.5	21.4	622	2	JC7973	synleurin - human
2	319.5	21.1	605	2	A41915	insulin-like growt
3	318	21.0	1469	2	B36665	slit protein 2 pre
4	318	21.0	1480	2	A36665	slit protein 1 pre
5	317	21.0	1531	2	T42218	slit-1 protein hom
6	313.5	20.7	605	2	JC5239	insulin-like growt
7	312	20.6	1523	2	T13953	MEGF5 protein - ra
8	288	19.1	603	2	JC6128	insulin-like growt
9	287	19.0	907	2	JE0176	orphan G protein-c
10	285	18.9	603	2	JC1282	insulin-like growt
11	282.5	18.7	560	2	A60164	platelet membrane

12	279.5	18.5	361	2	A53860	chondroadherin pre
13	278	18.4	907	2	JG0193	G protein-coupled
14	266	17.6	536	2	A34901	lysine carboxypept
15	253.5	16.8	1091	2	A58532	glial cell membran
16	249.5	16.5	420	2	A53531	oncofetal trophobl
17	243	16.1	312	1	NBHUA2	leucine-rich alpha
18	235	15.6	594	2	T23841	hypothetical prote
19	231.5	15.3	707	2	JC7763	neuronal leucine-r
20	230.5	15.3	359	1	NBHUC8	decorin precursor
21	229	15.2	682	2	A49121	cell-surface molec
22	229	15.2	682	2	A43318	connectin precurso
23	227.5	15.1	360	2	S06280	decorin precursor
24	227	15.0	610	2	T23836	hypothetical prote
25	221.5	14.7	360	2	I47020	decorin - rabbit
26	217.5	14.4	357	2	S24317	decorin precursor
27	214.5	14.2	354	2	A55454	decorin precursor
28	213	14.1	333	2	T34555	hypothetical prote
29	213	14.1	789	2	T28714	hypothetical prote
30	213	14.1	1355	2	T28715	hypothetical prote
31	209.5	13.9	1389	2	T13852	gene wheeler prote
32	207.5	13.7	1385	2	T13887	tlr protein - frui
33	207	13.7	562	2	T34319	hypothetical prote
34	207	13.7	653	2	T25194	hypothetical prote
35	204.5	13.5	738	2	T19938	hypothetical prote
36	204	13.5	382	2	I39068	proline- arginine-
37	203.5	13.5	354	2	S29145	decorin precursor
38	200.5	13.3	1112	2	T10504	disease resistance
39	200	13.2	1025	2	T42626	secreted leucine-r
40	199.5	13.2	1535	2	S46224	peroxidasin - frui
41	198.5	13.1	1066	2	T15864	hypothetical prote
42	196.5	13.0	375	2	S05390	fibromodulin precu
43	196	13.0	961	2	T23395	hypothetical prote
44	195.5	12.9	680	2	T19939	hypothetical prote
45	191.5	12.7	626	1	NBHUIA	platelet glycoprot

ALIGNMENTS

```
RESULT 1
JC7973
synleurin - human
C;Species: Homo sapiens (man)
C;Date: 25-Aug-2003 #sequence_revision 25-Aug-2003 #text_change 15-Sep-2003
C;Accession: JC7973
R;Wang, W.; Yang, Y.; Li, L.; Shi, Y.
Biochem. Biophys. Res. Commun. 305, 981-988, 2003
A;Title: Synleurin, a novel leucine-rich repeat protein that increases the intensity of pleiotropic
A;Reference number: JC7973; PMID:12767927
A;Accession: JC7973
A;Molecule type: mRNA
A;Residues: 1-622 <WAN>
A;Cross-references: GB:AY280614
C;Comment: This protein that is a single span transmembrane leucine-rich repeat protein is involved
C;Genetics:
A;Gene: slrn
A;Map position: 5q12.1
C;Keywords: cytokine; leucine-rich repeat; synleurin; transmembrane protein

  Query Match             21.4%;  Score 323.5;  DB 2;  Length 622;
  Best Local Similarity   27.1%;  Pred. No. 6.6e-21;
  Matches   96;  Conservative   41;  Mismatches  110;  Indels  107;  Gaps    5;

Qy         21 GLQAVPAGIPASSQRIFLHGNRISYVPAA----------------------SFQSCRN 56
              || ::|   | |:  ::| || |||:  :                      :|   |:
```

SCORE Search Results D

Score Home Page Retrieve Application List SCORE System Overview SCORE FAQ Comments / Suggestions

This page gives you Search Results detail for the Application 10553669 and Search Result 20071121_092712_us-1

```
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OM protein - protein search, using sw model

Run on:          November 21, 2007, 09:43:31 ; Search time 16 Seconds
                                        (without alignments)
                                        1938.665 Million cell updates/sec

Title:           US-10-553-669-6
Perfect score:   1695
Sequence:        1 CPGACVCYNEPKVTTSRPQQ.........TDEELLGLPKCCQPDAADKA 318

Scoring table:   BLOSUM62
                 Gapop 10.0 , Gapext 0.5

Searched:        283416 seqs, 96216763 residues

Total number of hits satisfying chosen parameters:      283416

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
                 Maximum Match 100%
                 Listing first 45 summaries

Database :        PIR_80:*
                 1:  pir1:*
                 2:  pir2:*
                 3:  pir3:*
                 4:  pir4:*

      Pred. No. is the number of results predicted by chance to have a
      score greater than or equal to the score of the result being printed,
      and is derived by analysis of the total score distribution.

                                SUMMARIES
```

Result No.	Score	% Query Match	Length	DB	ID	Description
1	328	19.4	622	2	JC7973	synleurin - human
2	321	18.9	1531	2	T42218	slit-1 protein hom
3	319.5	18.8	605	2	A41915	insulin-like growt
4	318	18.8	1469	2	B36665	slit protein 2 pre
5	318	18.8	1480	2	A36665	slit protein 1 pre
6	315	18.6	1523	2	T13953	MEGF5 protein - ra
7	313.5	18.5	605	2	JC5239	insulin-like growt
8	292	17.2	907	2	JE0176	orphan G protein-c
9	288.5	17.0	603	2	JC1282	insulin-like growt
10	288	17.0	603	2	JC6128	insulin-like growt
11	283	16.7	560	2	A60164	platelet membrane

12	279.5	16.5	361	2	A53860	chondroadherin pre
13	278	16.4	907	2	JG0193	G protein-coupled
14	266	15.7	536	2	A34901	lysine carboxypept
15	258.5	15.3	1091	2	A58532	glial cell membran
16	249.5	14.7	420	2	A53531	oncofetal trophobl
17	243	14.3	312	1	NBHUA2	leucine-rich alpha
18	235.5	13.9	594	2	T23841	hypothetical prote
19	232	13.7	707	2	JC7763	neuronal leucine-r
20	230.5	13.6	359	1	NBHUC8	decorin precursor
21	229	13.5	682	2	A49121	cell-surface molec
22	229	13.5	682	2	A43318	connectin precurso
23	227.5	13.4	360	2	S06280	decorin precursor
24	227.5	13.4	610	2	T23836	hypothetical prote
25	221.5	13.1	360	2	I47020	decorin - rabbit
26	217.5	12.8	357	2	S24317	decorin precursor
27	214.5	12.7	354	2	A55454	decorin precursor
28	213.5	12.6	653	2	T25194	hypothetical prote
29	213	12.6	333	2	T34555	hypothetical prote
30	213	12.6	789	2	T28714	hypothetical prote
31	213	12.6	1355	2	T28715	hypothetical prote
32	212	12.5	1389	2	T13852	gene wheeler prote
33	210	12.4	1385	2	T13887	tlr protein - frui
34	207	12.2	562	2	T34319	hypothetical prote
35	204.5	12.1	738	2	T19938	hypothetical prote
36	204	12.0	382	2	I39068	proline- arginine-
37	203.5	12.0	354	2	S29145	decorin precursor
38	203.5	12.0	1016	2	T30553	disease resistance
39	203.5	12.0	1535	2	S46224	peroxidasin - frui
40	203	12.0	1025	2	T42626	secreted leucine-r
41	202.5	11.9	1112	2	T10504	disease resistance
42	200.5	11.8	1066	2	T15864	hypothetical prote
43	196.5	11.6	375	2	S05390	fibromodulin precu
44	196	11.6	961	2	T23395	hypothetical prote
45	195.5	11.5	680	2	T19939	hypothetical prote

ALIGNMENTS

```
RESULT 1
JC7973
synleurin - human
C;Species: Homo sapiens (man)
C;Date: 25-Aug-2003 #sequence_revision 25-Aug-2003 #text_change 15-Sep-2003
C;Accession: JC7973
R;Wang, W.; Yang, Y.; Li, L.; Shi, Y.
Biochem. Biophys. Res. Commun. 305, 981-988, 2003
A;Title: Synleurin, a novel leucine-rich repeat protein that increases the intensity of pleiotropic
A;Reference number: JC7973; PMID:12767927
A;Accession: JC7973
A;Molecule type: mRNA
A;Residues: 1-622 <WAN>
A;Cross-references: GB:AY280614
C;Comment: This protein that is a single span transmembrane leucine-rich repeat protein is involved
C;Genetics:
A;Gene: slrn
A;Map position: 5q12.1
C;Keywords: cytokine; leucine-rich repeat; synleurin; transmembrane protein

  Query Match             19.4%;  Score 328;  DB 2;  Length 622;
  Best Local Similarity   26.6%;  Pred. No. 3.9e-21;
  Matches   98;  Conservative   45;  Mismatches  115;  Indels  110;  Gaps    6;

Qy         21 GLQAVPAGIPASSQRIFLHGNRISYVPAA----------------------SFQSCRN 56
              || ::|   | |:  ::| || |||:  :                      :|   |:
```

*tumor necrosis factor alpha inhibitor: AN, drug analysis
*tumor necrosis factor alpha inhibitor: DV, drug development
*tumor necrosis factor alpha inhibitor: DT, drug therapy
*tumor necrosis factor alpha inhibitor: TO, drug toxicity
*tumor necrosis factor alpha inhibitor: PO, oral drug administration
*tumor necrosis factor alpha inhibitor: PD, pharmacology
unclassified drug
unindexed drug
RN (amyloid beta protein) 109770-29-8; (calcium) 7440-70-2; (etanercept) 185243-69-0, 200013-86-1; (exendin 4) 141732-76-5, 141758-74-9; (glucagon like peptide 1) 89750-14-1; (infliximab) 170277-31-3; (pifithrin alpha) 63208-82-2; (thalidomide) 50-35-1

Full search history

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=> d his nofile

     (FILE 'HOME' ENTERED AT 10:36:57 ON 21 NOV 2007)

     FILE 'HCAPLUS' ENTERED AT 10:37:25 ON 21 NOV 2007
               E US20070065429 /PN
L1             1 SEA ABB=ON  PLU=ON  US20070065429 /PN
               D L1
               D SCAN

     FILE 'REGISTRY' ENTERED AT 10:39:02 ON 21 NOV 2007
L2             1 SEA ABB=ON  PLU=ON  786653-00-7/RN
L3             1 SEA ABB=ON  PLU=ON  786653-17-6/RN
L4             1 SEA ABB=ON  PLU=ON  786653-18-7/RN
L5             1 SEA ABB=ON  PLU=ON  786653-21-2/RN
L6             1 SEA ABB=ON  PLU=ON  786653-25-6/RN
L7             1 SEA ABB=ON  PLU=ON  783350-09-4/RN
L8             1 SEA ABB=ON  PLU=ON  783350-10-7/RN
L9             1 SEA ABB=ON  PLU=ON  783350-11-8/RN
L10            1 SEA ABB=ON  PLU=ON  783350-12-9/RN
L11            1 SEA ABB=ON  PLU=ON  783350-13-0/RN
L12            1 SEA ABB=ON  PLU=ON  783350-14-1/RN
L13            1 SEA ABB=ON  PLU=ON  783350-15-2/RN
L14            1 SEA ABB=ON  PLU=ON  783350-16-3/RN
L15            1 SEA ABB=ON  PLU=ON  783350-17-4/RN
L16            1 SEA ABB=ON  PLU=ON  783350-18-5/RN
L17            1 SEA ABB=ON  PLU=ON  783350-19-6/RN
L18            1 SEA ABB=ON  PLU=ON  783350-20-9/RN
L19            1 SEA ABB=ON  PLU=ON  783350-21-0/RN
L20            1 SEA ABB=ON  PLU=ON  783350-22-1/RN
L21            1 SEA ABB=ON  PLU=ON  783350-23-2/RN
L22            1 SEA ABB=ON  PLU=ON  783350-24-3/RN
L23            1 SEA ABB=ON  PLU=ON  790777-25-2/RN
L24            1 SEA ABB=ON  PLU=ON  790777-26-3/RN
L25            1 SEA ABB=ON  PLU=ON  790777-27-4/RN
L26            1 SEA ABB=ON  PLU=ON  790777-28-5/RN
L27            1 SEA ABB=ON  PLU=ON  790777-29-6/RN
L28            1 SEA ABB=ON  PLU=ON  790777-30-9/RN

     FILE 'HCAPLUS' ENTERED AT 10:48:08 ON 21 NOV 2007
L29            1 SEA ABB=ON  PLU=ON  L2
L30            1 SEA ABB=ON  PLU=ON  L3
L31            1 SEA ABB=ON  PLU=ON  L4
L32            1 SEA ABB=ON  PLU=ON  L5
L33            1 SEA ABB=ON  PLU=ON  L6
L34            3 SEA ABB=ON  PLU=ON  L7
L35            4 SEA ABB=ON  PLU=ON  L8
L36            4 SEA ABB=ON  PLU=ON  L9
L37            4 SEA ABB=ON  PLU=ON  L10
L38            4 SEA ABB=ON  PLU=ON  L11
L39            4 SEA ABB=ON  PLU=ON  L12
L40            4 SEA ABB=ON  PLU=ON  L13
L41            4 SEA ABB=ON  PLU=ON  L14
L42            4 SEA ABB=ON  PLU=ON  L15
L43            4 SEA ABB=ON  PLU=ON  L16
L44            4 SEA ABB=ON  PLU=ON  L17
L45            4 SEA ABB=ON  PLU=ON  L18
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L46 4 SEA ABB=ON PLU=ON L19
L47 4 SEA ABB=ON PLU=ON L20
L48 4 SEA ABB=ON PLU=ON L21
L49 4 SEA ABB=ON PLU=ON L22
L50 1 SEA ABB=ON PLU=ON L23
L51 1 SEA ABB=ON PLU=ON L24
L52 1 SEA ABB=ON PLU=ON L25
L53 1 SEA ABB=ON PLU=ON L26
L54 1 SEA ABB=ON PLU=ON L27
L55 1 SEA ABB=ON PLU=ON L28
L56 1 SEA ABB=ON PLU=ON (L29 OR L30 OR L31 OR L32 OR L33)
L57 4 SEA ABB=ON PLU=ON (L34 OR L35 OR L36 OR L37 OR L38)
L58 4 SEA ABB=ON PLU=ON (L39 OR L40 OR L41 OR L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49)
L59 1 SEA ABB=ON PLU=ON (L50 OR L51 OR L52 OR L53 OR L54 OR L55)
L60 4 SEA ABB=ON PLU=ON (L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35 OR L36 OR L37 OR L38 OR L39 OR L40 OR L41 OR L42 OR L43 OR L44 OR L45 OR L46 OR L47 OR L48 OR L49 OR L50 OR L51 OR L52 OR L53 OR L54 OR L55)
E ALZHEIMER
L61 47914 SEA ABB=ON PLU=ON (ALZHEIMER/BI OR ALZHEIMERS/BI)
L62 QUE ABB=ON PLU=ON ((NOGO OR NOGOR OR NOGOR1 OR NGR OR NGR1)(5A)(AGON? OR ANTAGON? OR RECEPT? OR PEPTID? OR POLYPEPT?))
L63 QUE ABB=ON PLU=ON ((NOGO OR NOGOR OR NOGOR1 OR NGR OR NGR1)(5A)(ALZHEIMER? OR ALSHEIMER? OR PLAQUE? OR AMYLOID? OR ALPHA? OR BETA?))
L64 39 SEA ABB=ON PLU=ON L61 AND L62
L65 19 SEA ABB=ON PLU=ON L61 AND L63
L66 3 SEA ABB=ON PLU=ON L60 AND L61
L67 4 SEA ABB=ON PLU=ON L60 AND L62
L68 2 SEA ABB=ON PLU=ON L60 AND L63
L69 QUE ABB=ON PLU=ON ((NOGO? OR NOGOR? OR NOGOR1? OR NGR? OR NGR1?)(5A)(ALZHEIMER? OR ALSHEIMER? OR PLAQUE? OR AMYLOID? OR ALPHA? OR BETA?))
L70 QUE ABB=ON PLU=ON ((NOGO? OR NOGOR? OR NOGOR1? OR NGR? OR NGR1?)(5A)(AGON? OR ANTAGON? OR RECEPT? OR PEPTID? OR POLYPEPT?))
L71 2 SEA ABB=ON PLU=ON L60 AND L69
L72 4 SEA ABB=ON PLU=ON L60 AND L70
L73 19 SEA ABB=ON PLU=ON L61 AND L69
L74 39 SEA ABB=ON PLU=ON L61 AND L70
L75 3 SEA ABB=ON PLU=ON L40 AND L61
L76 43 SEA ABB=ON PLU=ON L73 OR L74
L77 3 SEA ABB=ON PLU=ON L60 AND L61
L78 3 SEA ABB=ON PLU=ON (L71 OR L72 OR L77) AND L76
E AMYLOID/CT
L79 11765 SEA ABB=ON PLU=ON AMYLOID/CT
L80 1 SEA ABB=ON PLU=ON L60 AND L79
L81 7 SEA ABB=ON PLU=ON L79 AND L69
L82 10 SEA ABB=ON PLU=ON L79 AND L70
L83 12 SEA ABB=ON PLU=ON L81 OR L82
L84 44 SEA ABB=ON PLU=ON L83 OR L76
L85 QUE ABB=ON PLU=ON (RECEPT?(3A)(AGON? OR ANTAGON?))
L86 10 SEA ABB=ON PLU=ON L84 AND L85
L87 QUE ABB=ON PLU=ON ((PEPT? OR POLYPEPT?)(3A)(AGON? OR ANTAGON?))
L88 4 SEA ABB=ON PLU=ON L84 AND L87
L89 QUE ABB=ON PLU=ON ((ALPHA? OR ALFA? OR BETA?)(3A)(PEPTID? OR POLYPEPT? OR PROTE?))

L90 7129 SEA ABB=ON PLU=ON L79 AND L89
L91 QUE ABB=ON PLU=ON ((MAMMAL? OR PRIMAT? OR RODENT? OR DOG? OR CAT? OR PIG? OR RAT? OR MOUSE? OR MICE? OR HUMAN? OR MONKEY? OR PLACENT? OR MARSUP?)(3A)(BRAIN? OR CNS? OR (CENTRAL)(2A)(NERVOUS)))
L92 QUE ABB=ON PLU=ON (REDUC? OR ADMINIST? OR TREAT? OR ALLEV? OR AMELIOR? OR PALLIAT? OR PHARMAC? OR MEDICIN? OR MEDICAT? OR THERAP?)
L93 45 SEA ABB=ON PLU=ON (L71 OR L72 OR L73 OR L74) OR L77 OR (L80 OR L81 OR L82 OR L83 OR L84) OR L86 OR L88
L94 6 SEA ABB=ON PLU=ON L93 AND L90
L95 7 SEA ABB=ON PLU=ON L93 AND L91
L96 40 SEA ABB=ON PLU=ON L93 AND L92
L97 45 SEA ABB=ON PLU=ON (L93 OR L94 OR L95 OR L96)
L98 45 SEA ABB=ON PLU=ON L97 OR L77 OR L80
L99 QUE ABB=ON PLU=ON AY<2005 OR PY<2005 OR PRY<2005 OR REVIEW/DT

L100 23 SEA ABB=ON PLU=ON L98 AND L99
SAVE TEMP L100 HA669HCTX/A
E STRITTMATTER S?/AU
L101 144 SEA ABB=ON PLU=ON ("STRITTMATTER S M"/AU OR "STRITTMATTER STEPHEN"/AU OR "STRITTMATTER STEPHEN M"/AU OR "STRITTMATTER STEPHEN MARK"/AU OR "STRITTMATTER STEPHEN S"/AU)
E LEE D?/AU
L102 228 SEA ABB=ON PLU=ON "LEE DANIEL"?/AU OR "LEE DANIEL H S"?/AU OR "LEE D H S"?/AU OR "LEE DANIEL"/AU
E LI W?/AU
L103 4680 SEA ABB=ON PLU=ON "LI WEIWEI"/AU OR "LI WEI WEI"/AU OR "LI WEI"/AU OR "LI W W"/AU OR "LI WEI W"/AU
L104 5 SEA ABB=ON PLU=ON L101 AND L102 AND L103
L105 5038 SEA ABB=ON PLU=ON (L101 OR L102 OR L103)
L106 14 SEA ABB=ON PLU=ON L105 AND BIOGEN?/CO,CS,PA,SO
L107 97 SEA ABB=ON PLU=ON L105 AND YALE?/CO,CS,PA,SO
L108 78 SEA ABB=ON PLU=ON L107 AND L99
L109 30 SEA ABB=ON PLU=ON L108 AND (ALZHEIMER? OR ALSHEIMER? OR AMYLOID? OR PLAQUE? OR NOGO? OR NOGOR? OR NOGO1? OR NOGOR1? OR NGR? OR NGO? OR NGR1?)
L110 43 SEA ABB=ON PLU=ON L104 OR L106 OR L109
SAVE TEMP L110 HA669HCIN/A

FILE 'MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 11:40:06 ON 21 NOV 2007
L111 192138 SEA ABB=ON PLU=ON (ALZHEIMER? OR ALSHEIMER?)
L112 68782 SEA ABB=ON PLU=ON (AMYLOID?(4N)(PLAQUE? OR PEPTID? OR POLYPEPT? OR PROTEIN?))
L113 43928 SEA ABB=ON PLU=ON L111 AND L112
L114 0 SEA ABB=ON PLU=ON L113 AND L60
L115 10 SEA ABB=ON PLU=ON L113 AND L69
L116 17 SEA ABB=ON PLU=ON L113 AND L70
L117 18 SEA ABB=ON PLU=ON L115 OR L116
L118 862 SEA ABB=ON PLU=ON L113 AND L85
L119 62 SEA ABB=ON PLU=ON L113 AND L87
L120 34573 SEA ABB=ON PLU=ON L113 AND L89
L121 23 SEA ABB=ON PLU=ON L118 AND L119 AND L120
L122 787 SEA ABB=ON PLU=ON (L118 OR L119) AND L92
L123 41 SEA ABB=ON PLU=ON L117 OR L121
L124 6 SEA ABB=ON PLU=ON L123 AND L91
L125 32 SEA ABB=ON PLU=ON L123 AND L92
L126 32 SEA ABB=ON PLU=ON L124 OR L125
L127 41 SEA ABB=ON PLU=ON L123 OR L126
L128 21 SEA ABB=ON PLU=ON L127 AND L99

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L129          6 SEA ABB=ON  PLU=ON  L117 AND L128
L130          6 SEA ABB=ON  PLU=ON  L117 AND L99
L131         21 SEA ABB=ON  PLU=ON  L128 OR L129 OR L130
               SAVE TEMP L131 HA669MLTX/A
L132          4 SEA ABB=ON  PLU=ON  "NOGO RECEPTOR ANTAGONIST?"
L133          0 SEA ABB=ON  PLU=ON  L131 AND L132
L134          1 SEA ABB=ON  PLU=ON  L132 AND L99
               D SCAN
L135         29 SEA ABB=ON  PLU=ON  (NOGO(4N) RECEPTOR) AND (AMYLOID? OR
               ALZHEIMER? OR ALSHEIMER?)
L136         20 SEA ABB=ON  PLU=ON  L135 AND L92
L137          0 SEA ABB=ON  PLU=ON  (NGO(4N) RECEPTOR) AND (AMYLOID? OR
               ALZHEIMER? OR ALSHEIMER?)
L138         16 SEA ABB=ON  PLU=ON  (NGR(4N) RECEPTOR) AND (AMYLOID? OR
               ALZHEIMER? OR ALSHEIMER?)
L139          0 SEA ABB=ON  PLU=ON  (NOGOR(4N) RECEPTOR) AND (AMYLOID? OR
               ALZHEIMER? OR ALSHEIMER?)
L140         13 SEA ABB=ON  PLU=ON  L112 AND L69
L141         23 SEA ABB=ON  PLU=ON  L112 AND L70
L142         26 SEA ABB=ON  PLU=ON  (L140 OR L141)
L143         33 SEA ABB=ON  PLU=ON  L135 OR (L137 OR L138 OR L139) OR L142
L144         24 SEA ABB=ON  PLU=ON  L143 AND L92
L145          5 SEA ABB=ON  PLU=ON  L144 AND L99
L146         23 SEA ABB=ON  PLU=ON  L145 OR L131
               SAVE TEMP L146 HA660MLTX/A
L147          6 SEA ABB=ON  PLU=ON  L104
L148         27 SEA ABB=ON  PLU=ON  L106
L149         20 SEA ABB=ON  PLU=ON  L148 AND L99
L150         24 SEA ABB=ON  PLU=ON  L147 OR L149
               SAVE TEMP L150 HA669MLIN/A
               D QUE L110
               D QUE L150

     FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, DRUGU' ENTERED AT 12:07:44 ON 21
     NOV 2007
L151         50 DUP REM L110 L150 (17 DUPLICATES REMOVED)
                     ANSWERS '1-43' FROM FILE HCAPLUS
                     ANSWERS '44-49' FROM FILE BIOSIS
                     ANSWER '50' FROM FILE DRUGU
               D L151 1-50 IBIB AB
               D QUE L100
               D QUE L146
L152         36 DUP REM L100 L146 (10 DUPLICATES REMOVED)
                     ANSWERS '1-23' FROM FILE HCAPLUS
                     ANSWERS '24-29' FROM FILE MEDLINE
                     ANSWERS '30-32' FROM FILE BIOSIS
                     ANSWERS '33-36' FROM FILE EMBASE
               D L152 1-23 IBIB ED ABS HITIND
               D L152 24-36 IBIB AB HIT
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